LII.

THE ROENTGEN RAY AS AN AID TO THE DIAGNOSIS OF DISEASES OF THE SPHENOID SINUS.*

BY ROSS HALL SKILLERN, M. D.,

AND

GEORGE E. PFAHLER, M. D.,

PHILADELPHIA.

(DR. SKILLERN.) The diagnosis of sphenoidal sinusitis at best is not an easy matter, and even in the frank cases with pus appearing both in the olfactory fissure and nasopharynx, it often requires repeated examinations before we can trace the purulent secretion to its origin. The reason for this lies of course in the anatomic configuration of the parts. The ostium of the sphenoid lying usually deep in the spheno-ethmoidal recess, and this cavity being concealed anteriorly by the superior and middle turbinates, a resection of one or both of these structures is demanded before the anterior sphenoidal wall is accessible to the eye, and subsequently to instrumentation.

A great many cases of sphenoiditis occur in which the general symptoms are marked, but local symptoms are mild or even fail entirely, at least at the time of examination. Only the merest traces of pus can be found, even on painstaking inspection, and certainly not enough to warrant the removal of practically the entire posterior ethmoid labyrinth in order to arrive at a conclusion. While in a given case we are more or less convinced in our own minds that the mucosa of the sphenoid is inflamed, we do not feel that the general symptoms justify the exploratory exenteration of the posterior ethmoid cells with resection of a portion of the anterior sphenoidal wall. Here is when the long bladed Killian speculum serves a useful purpose. Introduced between the middle turbinate and septum, after thorough cocainization of these, the blades are gradually sprung apart until the recessus sphenoethmoidalis is visible. As the distance between the blades is often not more than one-eighth of an inch, it is frequently necessary to swab away the blood and secretion which accumulates between them until a comparatively dry field is obtained.

It has been our experience in these cases (where pus is not exuding from the ostium) that the opening into the sphenoid sinus is not readily found, despite the fact that an untoward amount of secretion is present in the sphenoethmoidal region. We are now in the position of being morally certain that inflammatory changes are present in the mucosa of the sphenoid, but are unable to prove it, owing to our inability to find the ostium, even with the sound. Before applying any actual surgical intervention we have one means more at our command—the negative pressure or suction apparatus of Sonderman.

I use a simplified form of the apparatus, which consists merely of a modified glass tube, closed with a rubber cork, one end of which the patient holds in the nostril, the other end being attached to a four-ounce metal syringe. After causing the patient to close both nostrils tightly with the thumb and finger so that no air can leak out around the tube, he is told to articulate a long E—thus closing the nasopharynx with the uvula and soft palate. The piston of the syringe is sharply pulled to the end of the barrel, thus creating a vacuum in the nares on that side, the extent of which depends upon the force with which the piston is retracted as well as upon the leakage

of air around the glass tube. This maneuver is repeated a number of times and the sphenoethmoidal region again inspected, using the long Killian speculum.

If a decided increase in the quantity of secretion is present, the probabilities are that much greater of the presence of sphenoidal disease. Suppose, however, the ostium still remains invisible, are we justified in opening the anterior wall in order to arrive at a diagnosis? That depends entirely upon the severity of the general symptoms, and as in these cases the symptoms are for the most part moderate, this answer should be in the negative. If a sound can be made to penetrate the ostium, even though the opening be concealed in the depths of the ethmoidal recess, it is perfectly justifiable to introduce a cotton carrier saturated with a strong solution of cocain and adrenalin and endeavor to dilate the mucosa which covers the bony opening in the form of a diaphragm, for should we succeed in this, a much better condition is made for the outflow of any secretion which might lie therein.

I have observed time after time that when the cotton tipped carrier was introduced into, but not completely through, the ostium, allowed to remain about five minutes, and then while the patient bent the head forward until the chin rested on the chest, if the instrument was suddenly removed, purulent secretion in no inconsiderable amount would follow and be plainly visible on the anterior sphenoidal wall. We consider this when present as absolutely diagnostic of sphenoidal sinusitis, in spite of the fact that the ostium remains hidden from view.

Suppose, however, it was impossible to find the ostium with the sound and there remained only the alternative of creating an artificial opening in the anterior wall. At this point the X-ray finds its first great period of usefulness, for with the modernized technic reliable deductions can be made from negatives taken in proper positions. From four to six exposures in different angles should be made of each case in order to thoroughly differentiate the existing conditions. From a rhinologist's point of view the important points elicited are as follows: 1. The difference in the shadow intensity of the two sides. 2. Whether the shadow extends into the posterior ethmoid cells. 3. The size of the sphenoid sinus. 4. The distance between the anterior sphenoidal wall and the vestibulum nari. 5. The shape of the sphenoid sinus with especial

reference to the posterior or cerebral wall. All these points are of the utmost importance; the first and second for diagnostic, and the third, fourth and fifth for operative purposes. As this brings us to the province of the roentgenologist, I take pleasure in presenting to the fellows my coworker in the subject, Dr. George Pfahler.

ROENTGEN RAYS.

(DR. PFAHLER.) Dr. Skillern has set forth the needs of aid in the diagnosis of the sphenoidal sinuses. You as expert rhinologists know this need better than I can tell, therefore I shall confine my remarks to a very brief review of what has been accomplished in this line of research, describe what I have found to be a satisfactory technic, and review what I believe to be the possibilities.

The recognition of disease of the sphenoidal sinuses by means of the Roentgen rays follows naturally the study of diseases of the other sinuses and involves only an amplification or refinement in the technic which has so successfully demonstrated disease in the other accessory sinuses. Therefore, all of the long list of investigators who have contributed to the study of the accessory sinuses in general have paved the way for the demonstration of disease in the sphenoidal cells, and for this reason are included in the bibliography.

The general impression among rhinologists and roentgenologists is that the rays are of little value in demonstrating disease of these sinuses. This impression must arise either from faulty technic or from lack of skill in interpretation of the negatives that were made; and I believe the chief fault has been lack in interpretation. I find among my negatives made six and eight years ago distinct evidence of sphenoidal disease which I had not recognized.

Special work has been done along this line by Pfeiffler, Spie, Scheier, Rhese, Bertini, Brunglow, Marseblick and Schuller. Rhese has recorded more particularly the end results of the roentgenologic studies. In his 68 cases with roentgenologic studies, 77 ethmoid labyrinths were found diseased in 53 patients, which were confirmed by operation. Among these cases the roentgenogram showed no evidence of disease in two, in which disease was found at operation.

In two others the evidence of disease was only slight, as shown by the roentgenogram.

In 31 patients who came for operation, 40 sphenoidal sinuses were found diseased. Of these, four were not recognized by the Roentgen rays. In none of these cases in which the disease was unrecognized was there a complete examination. Rhese lays great stress on the importance of the oblique view, and concludes that where a posteroanterior and oblique view show no evidence of disease, there should be no extensive operation undertaken.

He also calls attention to the fact that among the cases studied, four showed disease roentgenologically which was not found at operation. He concludes that previous disease probably resulted in granulation tissue which gave a cloudy appearance to the cells.

TECHNIC.

In the first place, I believe we should always make our studies complete, and I believe that a complete study of the sphenoid involves the other accessory sinuses as well. This of course increases the time consumed as well as the expense, but it will go far toward eliminating error, and will at the same time, I am sure, often show more extensive disease than would otherwise be expected, and by so doing the rhinologist is given full information concerning all of the sinuses, and can treat at once all of them more skillfully, thus lessening the duration of the illness and in the end being distinctly economical.

In my judgment a complete study involves two posteroanterior exposures, two lateral (made stereoscopically) and two oblique. In certain cases to this might be added an anteroposterior and a vertical. When the exposures must be limited as much as possible, either on account of the expense or condition of the patient, one posteroanterior and one lateral view will give most information and in most instances will suffice for a diagnosis.

The posteroanterior view (No. 1) is made by placing the patient in a prone position. (When circumstances actually demand it, the patient may be allowed to sit up or may lie upon his back, but this will involve unusual or improvised appliances.) The plate is placed under the face so as to in-

clude the frontal sinuses and the maxillary sinuses. The tube is placed over the occipital region at a distance of twenty-two inches and located so that the central ray will pass through the median line and in a posteroanterior plane which extends from the base of the nose through the external auditory meatus. This position must be exact, otherwise if the tube is placed too high, the area of the sphenoidal sinus will be projected downward over the middle turbinated bones, and if too low they will be thrown over the shadow of the cribriform plate of the ethmoid.

If the proper position has been used and a good plate obtained, the reading of the negative from above downward will be as follows: (1) Frontal bone and brain tissue; (2) frontal sinuses, not well shown; (3) cribriform plate with a few small ethmoidal cells below; (4) on each side of the median line (in a normal skull) a triangular transparent area with rounded corners, the base of the triangle being in the median line or septum. These areas are approximately one-half to three-fourths of an inch in diameter. If they are diseased they will be opaque in proportion to the amount of exudate or tumor tissue contained. They may be irregular, deformed, or absent. Their walls may be broken down by disease (such as syphilis), or they may be excessively dense as from osteoma; (5) on either side of the sphenoidal cells one will see the projected shadows of the posterior ethmoidal cells and below them the anterior ethmoidal cells; (6) below this area are the nasal cavities with the middle turbinate above and the inferior turbinate below; (7) on either side of the nasal cavities are the large triangular transparent areas of the maxillary sinuses; (8) projected into these sinuses one sees the shadow of the atlas bone.

Posteroanterior position (No. 2) is made particularly to demonstrate the frontal sinuses and thereby give confirmatory or additional information. For this the patient is kept in the same position, the tube is kept in the median line, but is moved about two inches above the occiput. This will vary somewhat with the shape of the head. The chief features in this negative will be the frontal sinuses above, and the lateral ethmoid cells below, lying just above the upper orbital brim. Below this are found the structures enumerated under position No. 1.

The oblique position is used for each side, and both sides should be made for comparison. The patient's head is placed so that the brim of the orbit is resting upon the plate. This will make the weight of the head rest upon the superciliary ridge, the nose and malar bone. The tube is placed at a distance of 22 inches from the plate and in such a position that the central ray will enter the opposite parietal region about two inches posteriorly and one and one-half inches above the external auditory meatus and projected toward the center of the orbit.

If this plate is properly made the optic foramen will occupy the center of the orbit, and to the outer side will be found the sphenoidal fissure. Then toward the median line will be projected the sphenoidal sinus—anterior to the optic foramen. Above this will be seen the upper brim of the orbit, and above the orbit the frontal sinuses.

The lateral view should be made stereoscopically, when possible, because it will give clearer and more definite information concerning the sphenoidal sinuses, the ethmoidal cells and the sella turcica above.

When only one exposure is made the supposed affected side of the skull should be placed upon the plate. The tube is located at a distance of twenty-two inches so that the normal central ray will pass through the middle of a line drawn from the external auditory meatus and directed perpendicularly toward the plate. When stereoscopic plates are made this same central position should be found, and then the tube should be moved one and one-fourth inches anteriorly or posteriorly from this central point.

In the negatives or stereoscopic views one will then see posteriorly (1) the external auditory meatus, and anteriorly to this (2) the temporomaxillary articulation; continuing forward from this point is (3) the zygomatic arch, and above the zygoma are (4) the sphenoidal sinuses. The sphenoidal sinuses are bounded above by the sella turcica, below by the zygoma, posteriorly by the petrous portion of the temporal bone, and anteriorly by the anterior wall of the sinus; anteriorly to the sphenoid one recognizes (5) the posterior ethmoidal cells; (6) the anterior ethmoidal cells, and above and anteriorly to these cells (7) the frontal sinuses; through the ethmoidal cells can be traced the outline of (8) the orbit, and below the orbit (9) the maxillary sinus.

The anteroposterior view is used in exceptional instances—when on account of the physical or mental condition the patient is neither able to sit up nor lie upon the face, such as would occur in fracture of the base of the skull, abscess of the brain, or marked dyspnea. This position has been recommended by Marseblick and Schuller. The patient is placed upon his back, with his occiput upon the plate. The tube is then centered over the base of the nose. This will project the transparent areas of the sphenoid through the occipital region and the outlines of the orbits. An opacity of one or both of these areas, in a plate which showed good detail elsewhere, would indicate disease or exudate in one or both sinuses.

<div align="center">REMARKS.</div>

The various accessory sinuses are recognized by the greater degree of transparency as compared with the surrounding tissues. Generally disease of these sinuses is associated with an exudate which will diminish the transparency. When the sinus of one side only is involved, it forms a distinct contrast with the opposite side, which should be clear. If both sides are involved, this fact can be recognized, providing the negative is good. A good negative will show clear lines elsewhere, even though the sphenoidal area may be quite opaque and all lines may be obliterated.

If both sides seem to be diseased, as is indicated in the posteroanterior plate, this fact can be more positively shown by the opacity of the sphenoidal sinus, as is shown in the lateral or stereoscopic views.

Not only are the rays useful in diagnosing catarrhal and purulent disease of the sphenoidal sinuses, but valuable information will be obtained also in demonstrating the extent of new growths, and thus complete operation can be done when advisable, or a meddlesome operation may be avoided. (Marschick and Schuller have studied three cases of malignant disease of the sphenoidal sinus in which the floor of the sella turcica was absorbed.) Such new growths will include carcinoma, sarcoma, osteoma, exostosis, fibroma, cysts and mucocele. Infections such as tuberculosis and lues may be studied.

Other indications for a roentgenologic examination are to determine (1) the variety of the sinuses, whether to determine

the absence of normal sinuses or to determine abnormal pneumatic spaces (recessus supraorbitalis and hollow septum nasi) ; (2) accidents to the skull—foreign bodies, fractures or collections of blood in the sinus resulting from fractures at the base of the skull; (3) tumors of the accessory sinuses as well as of the base of the skull; (4) intracranial disease, especially hypophyseal tumors.

CONCLUSIONS.

1. The demonstration of diseases of the sphenoidal sinus by means of the Roentgen rays depends upon good technic and good negatives. Fluoroscopy need not be considered.

2. As much or more will depend upon the proper interpretation of the negatives as in the making of them.

3. The examination should include all of the sinuses and in each instance should be thorough. This will involve at least four different exposures, and usually six or more.

4. Under the above conditions we believe that inflammations (catarrhal or purulent), infections (tubercular, luetic), and new growths (benign and malignant) can be diagnosed in most if not all instances with great accuracy.

BIBLIOGRAPHY.

Beck, Joseph: Photographic Atlas of Radiography of the Mastoid Region and of the Nasal Accessory Sinuses. St. Louis. 1911.

Bertini: Anatomia radiografica sui seni sfenoidali. Torino, 1911.

Bornhaupt: Ein Fall von linksseitigem Stirnhöhlenosteom. Langenbeck Archiv., 1881, Bd. XXVI.

Brünings: Neue röntgenographische Darstellungamethode der Nebenhöhlen und Schläfebeine. Verhandlungen deutscher Laryngologen, Dresden, 11 u. 12, May, 1910.

Brunzlow: Die Darstellung der Nasenhöhlen und ihrer Erkrankungen im Röntgenbild. Fortschritte auf dem Gebiete der Röntgenstrahlen, 1911, Band 17, Heft 4.

Burger: Die Bedeutung der Röntgenstrahlen in der Rhinolaryngologie. Referat auf dem 1. internationalen Rhinolaryngologen-Kongress, Wien, 1908. (Als Monographie erschienen unter dem Titel: Was leisten die Röntgenstrahlen auf dem Gebiete der Rhinolaryngologie? 1908.)

Caldwell: Skiagraphy of the Accessory Sinuses of the Nose. American Quarterly of Roentgenology, January, 1907.

Chiari: Eine Modifikation der Schlofferschen Operation bei Hypophysentumoren. Wiener klin. Wochenschrift, 1912, No. 1.

Chiari und Marschik: Nasensarkom, Differentialdiagnose mittels Röntgenstrahlen. Annales des maladies de l'oreille, 1907.

858   ROSS HALL SKILLERN AND GEORGE E. PFAHLER.

Ciegynski, A.: Ueber extrakranielle Kieferaufnahmen mittels Röntgenstrahlen. Fortschritte auf dem Gebiete der Röntgenstrahlen, 1911, Heft 1, Band 18.

Cryer: Use of the Roentgen Rays in the Studies of the Normal and Pathologic Anatomy of the Internal Structures of the Face. Amer. Jour. of Medical Science, February, 1905.

Destot: Röntgenbilder bei Sinusitis maxillaris. Soc. de chirurgie de Lyon, 22, March, 1906.

Fabiunke: Beiträge zur Röntgendiagnostik der Mund und Kieferhohle. Berliner klinische Wochenschrift, 1909, p. 571.

Fischer und Hald: Röntgenuntersuchung der Nasennebenhöhlen. Ref. Zentralblatt für Ohrenheilkunde, 1908.

Goldmann und Killian: Ueber die Verwendung der X-Strahlen für die Bestimmung der nasalen Nebenhöhlen und ihrer Erkrankungen. Tübingen, 1907.

Haenel, F.: Osteombildung in samtlichen Nebenhöhlen der Nase. Gesellschaft für Natur- und Heilkunde, Dresden, 20, February, 1904.

Hajek: Pathologie und Therapie der entzündlichen Erkrankungen der Nebenhöhlen der Nase, 1909.

Hirsch, O.: Ueber Methoden der operativen Behandlung von Hypophysistumoren auf endonasalem wege. Archiv. für Laryngologie, Band 24, Heft 1.

Jansen: Was leistet das Röntgenverfahren auf otiatrischem Gebiete für die Diagnose. Deutsche Zeitschrift für Chirurgie, 1909.

Jungherr: Die bisherigen Leistungen der Röntgenphotographie auf dem Gebiete der Rhino-Laryngo- und Otologie. Zeitschrift für Elektrologie und Röntgenkunde, Band 9, Heft 4-7.

Kahler: Ein uberzahliger Zahn in der Nase. Wiener klinische Wochenschrift, 1905, No. 40.

Killian: Die Röntgenphotographie in Dienste der Rhinologie. 1. inter. laryn. rhin. Kongress, Wien, 1908.

Kirchhoff: Osteom der Stirnhöhle. Inaugural dissertation, Bonn, 1907.

Krotoschiner: Radiogramm der Nasennebenhöhlen. Breslauer chirurgische Gesellschaft, 12, July, 1909. Ref. Berliner klinische Wochenschrift, 1909, p. 1672.

Kuttner: Die entzündlichen Nebenhöhlen erkrankungen der Nase im Röntgenbilde. 1908.

Marschik und Schuller: Beitrag zur Röntgendiagnostik der Nebenhöhlen-erkrankungen. Fortschritte auf dem Gebiete der Röntgenstrahlen, Band 28, No. 4, 1912.

Onodi: Röntgenaufnahmen der Stirnhöhlen. Verhandlungen das 1. inter. Laryngologen Kongress. Wien, 1909.

Peckert: Das Antrum in Röntgenbild. Deutsche zahnärztliche Wochenschrift, 1907, No. 16.

Perthes: Die Bedeutung dur Röntgenstrahlen für die Diagnose und Operation der Stirnhöhlenosteome. Archiv. für klinische Chirurgie, Band 72, Heft 6.

Peyser: Die Röntgenuntersuchung der Nasennebenhöhlen. Archiv. für Laryngologie, Band 21, Heft 1.

Pfahler: Die isolierte Aufnahme einer Oberkieferhalfte und die isolierte Aufnahme des Processus styloideus. Fortschritte auf dem Gebiete Röntgenstrahlen, Band 17, Heft 6.

Pfeiffer: Eine neue Röntgenographische Darstellungsmethode der Keilbeinhöhlen. Archiv. für Laryngologie und Rhinologie, Band 23, p. 420-428, 1910.

Philip: The X-rays in Determining the Limits of the Frontal Sinus. Jour. of Amer. Med. Assoc., 1902.

Preysing: Spongiosierung der Stirnhöhlen. Zeitschrift für Laryngologie, Rhinologie und ihre Grenzgebiete, 1910, Band 3, Heft 4.

Reiner: In die Nasenhöhle aberrierter Zahn. Berliner laryngologische Gesellschaft, 21, May, 1909.

Reynier and Glover: Radiographic Researches on the Topographic Relations of the Brain, the Frontal and Maxillary Sinuses and the Venous Sinuses of the Dura Mater to the Walls of the Skull. Lancet, 1900.

Rhese: Die Diagnostik der Erkrankungen des Siebbeinlabyrinthes und der Keilbeinhöhlen durch das Röntgenverfahren. Deutsche medizinische Wochenschrift, 1910.

Rhese: Die chronischen Entzündungen der Seibbeinzellen und der Keilbeinhöhlen mit besonderer Berucksichtigung ihrer Beziehungen zur allgemeinen Medizin und ihrer Diagnostik durch das Röntgenverfahren. Archiv. für Laryngologie und Rhinologie, Band 24, 1911.

Scheier: Ueber die Verwertung der Röntgenstrahlen in der Rhino- und Laryngologie. Archiv. für Laryngologie, Band 6.

Scheier: Die Diagnostik der Empyeme der Nasennebenhöhlen und das Röntgenverfahren. Archiv. für Laryngologie und Rhinologie, 1909, Band 21, Heft 3.

Scheier: Zur Untersuchung der Keilbeinhöhlen mittels Röntgenstrahlen. Berliner klinische Wochenschrift, 1911, p. 37. Archiv. für Laryngologie und Rhinologie, Band 24, 1911.

Spiess: Die Röntgenuntersuchung der oberen Luftwege. Atlas von Groedel, 1909.

Underwood: An Inquiry Into the Anatomy and Pathology of the Maxillary Sinus. Jour. of Anatomy and Physiology, 1910, Vol. XLIV.

Uyeno: Das Osteofibrom des Oberkiefers, eine typische Geschwulst. Beitrage zur klinischen Chirurgie, Band 65, December, 1909.

Voss, F.: Das Sarcom des Keilbeines, ein typisches Geschwulst-Krankheitsbild. St. Petersburger medicinische Wochenschrift, 1910, p. 205.

Wassermann: Die Bedeutung des Röntgenverfahrens für die Diagnose Siebbein-und Stirnhöhleneiterungen. 16. Versammlung der otologischen Gesellschaft, Bremen, 1907. Arztlicher Verein, Munchen, June, 1907, und Internationaler laryngo-rhinologischer Kongress, Wien, 1908.

Ibid.: Die Bedeutung des Röntgenverfahrens auf dem Gebiete der Rhinologie und Laryngologie. Fortschritte auf dem Gebiete der Röntgenstrahlen, 1908.

Ibid.: Sammelreferat. Centralblatt für Ohrenheilkunde, 1907, p. 429.

Watsch: Stereoskopische Skiagramme der Nebenhöhlen. Londoner laryngol. Gesellschaft, Janner, 1907.

Weil: Röntgenaufnahmen der Nebenhöhlen nach Injektion von schattengebenden Massen. Wiener klinische Wochenschrift, 1903, p. 1471, und 1904, No. 2, p. 61.

Ibid.: Röntgenbild mit dem Nachweis der Kommunnikation beider Stirnhöhlen. Wiener laryngologische Gesellschaft, May, 1908.   Ref. Wiener klinische Wochenschrift, 1908, No. 34, p. 1893.

Wiegmann: Ein Fall von Osteom des Siebbeines. Zeitschrift für Ohrenheilkunde und Krankheiten der Luftwege, Band 62, Heft 1.

Winkler: Eitrige Erkrankungen der oberen nasalen Nebenraume des Gesichtsschädels. Fortschritte auf dem Gebeite der Röntgenstrahlen, Band 6, p. 79.

Ibid.: Falle von nasalen Nebenhöhlenerkrankungen. 73 Naturforscherversammlung in Hamburg, 1901.

Ibid.: Die Orientierung auf dem Röntgenbild des Gesichtsschädels und das Studium der oberen Nasennebenhöhlen auf demselben. Fortschritte auf dem Gebeite der Röntgenstrahlen, Band 5, p. 147.

Ibid.: In welcher Weiss kann bei eitrigen erkrankungen der oberen Nasennebenraume das Röntgenbild des Gesichstsschädels den operationsplan, dieses Hohlraume durch aussere Eingriffe freizulegen, modifizieren? Fortschritte auf dem Gebeite der Röntgenstrahlen, Band 6.

Witt: Ausbreitung der Stirnhöhlen und Siebbeinzellen über die Orbita.   Anatomische, Heft 1908, Band 37, Abtg. 1.
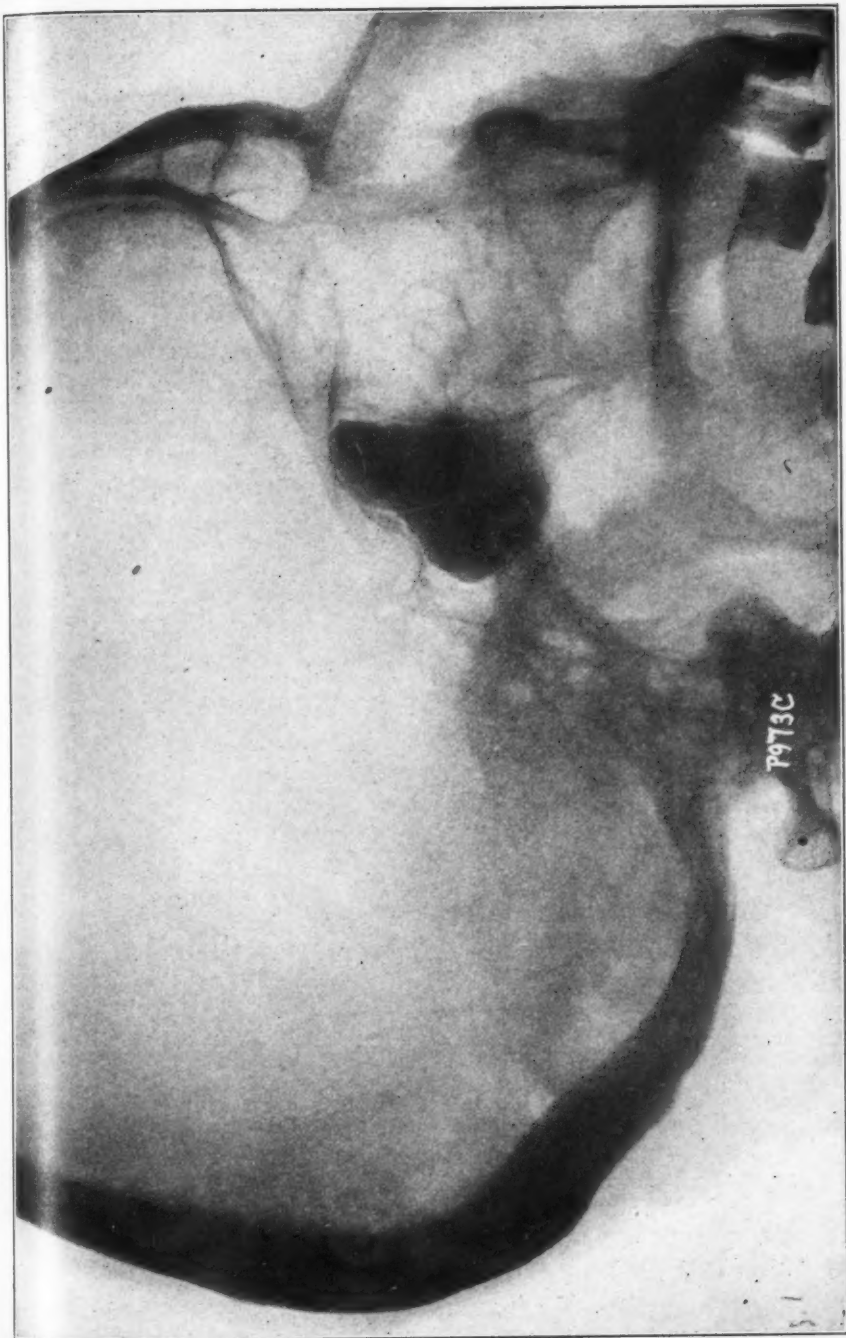
FIGURE 1.

Shows lateral view of a skull in a cadaver, with the right sphenoidal sinus injected with bismuth paste. Notice that the left sphenoidal is larger than the right and projects beyond it posteriorly.
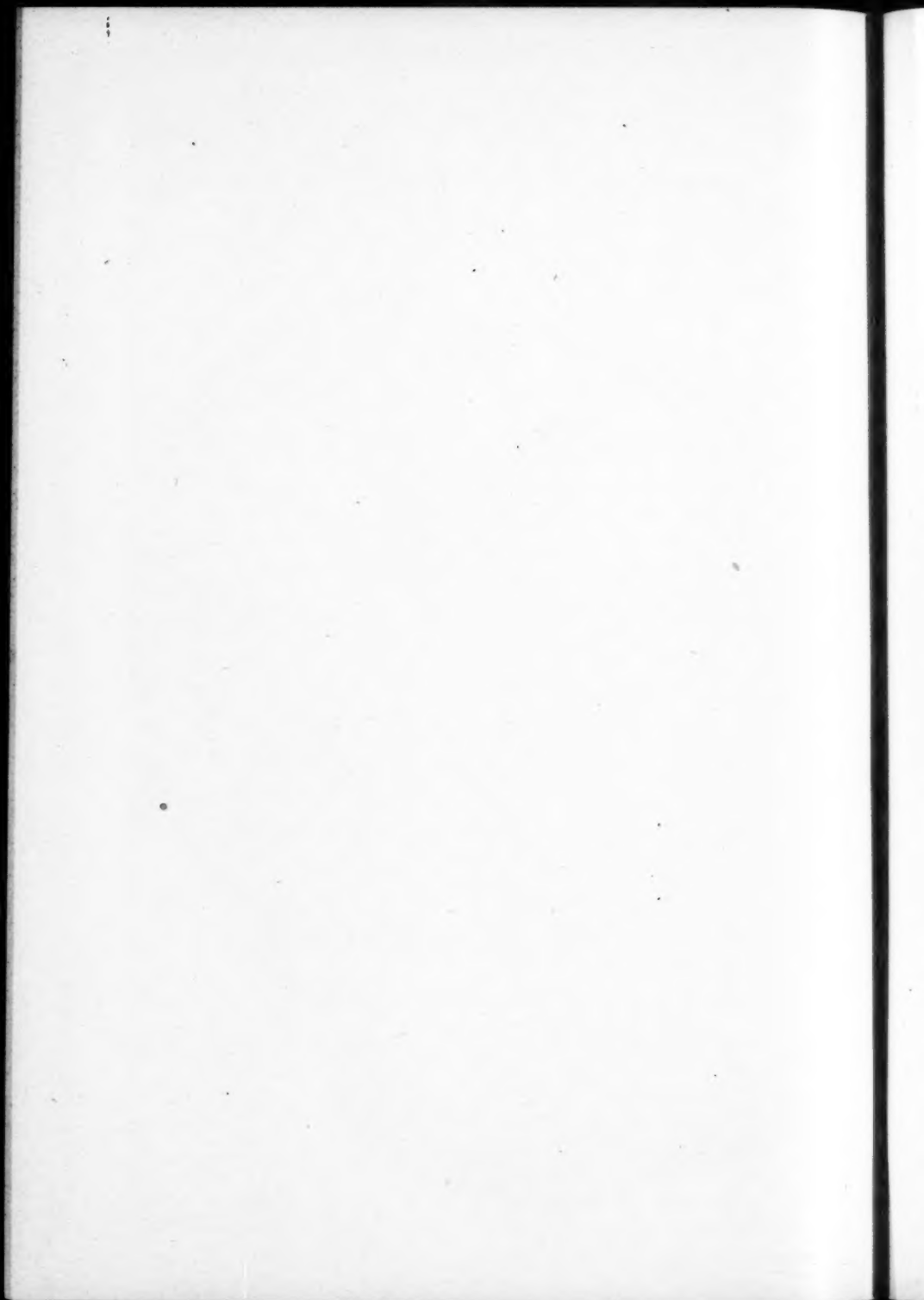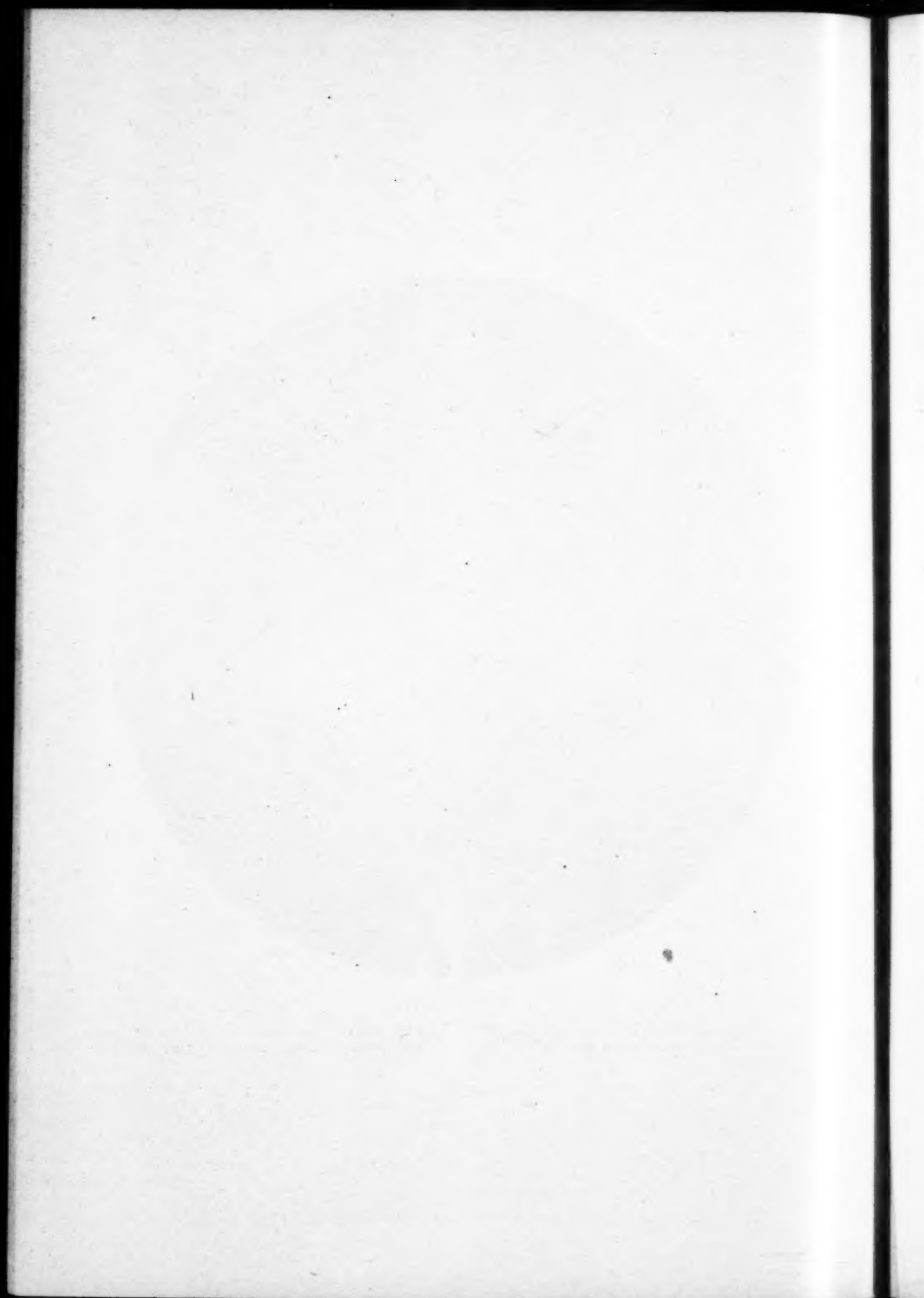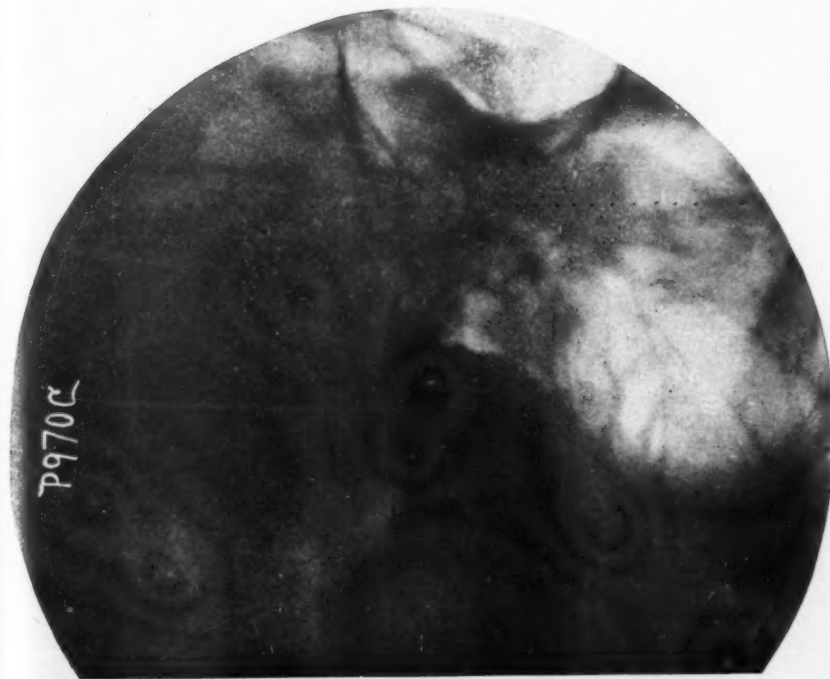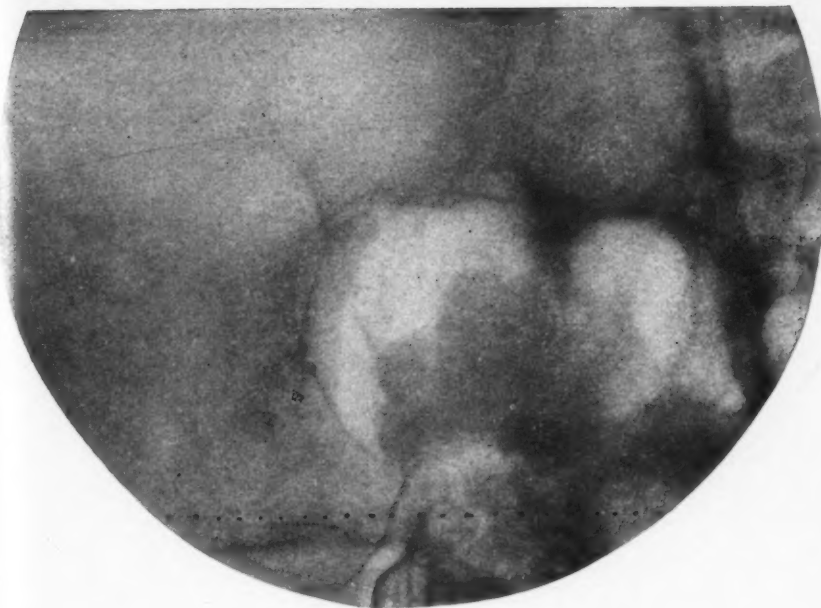
FIGURE II.

Right sphenoidal sinus injected with bismuth paste in a cadaver. Posteroanterior view. Notice the dotted outline of the left sinus, which shows as clearly as the one injected.
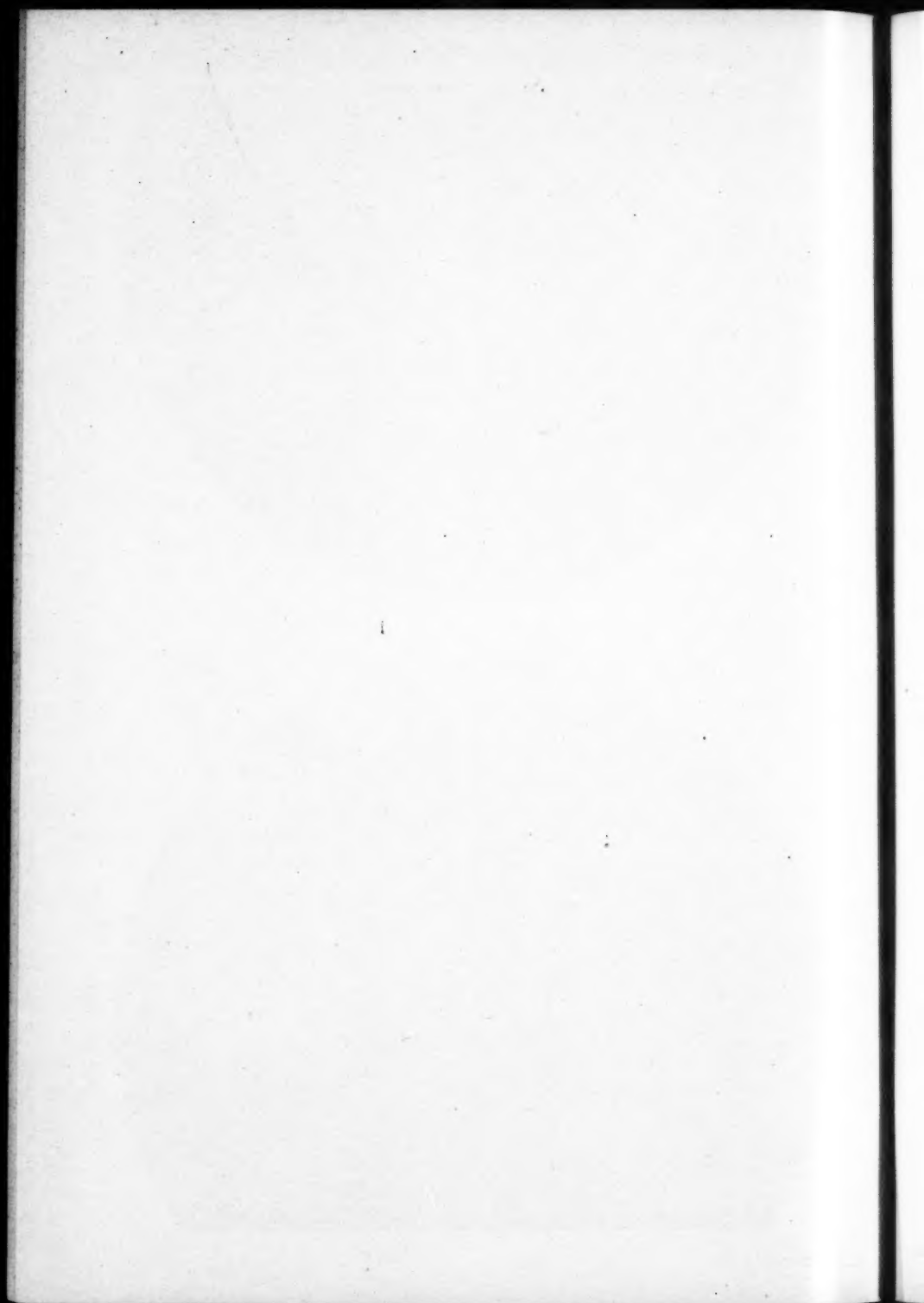
Right Oblique.　　　　　　　　　　　　　　　　Left Oblique.

FIGURE III.

Right and left oblique view of injected right sphenoidal sinus. Notice how much further the injected sinus is thrown from the median line in the left oblique view the left sinus, which is thrown into the outer side of the left orbit, is perfectly clear. In the right oblique view.
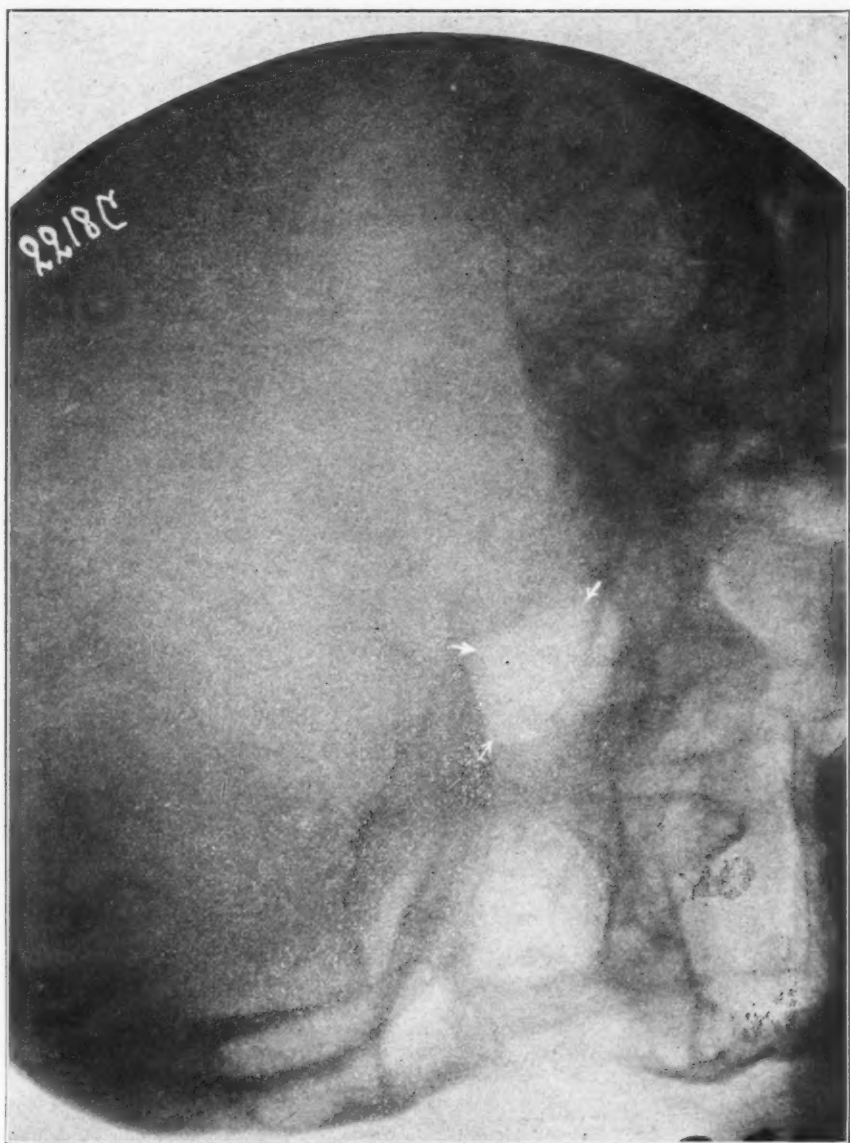
FIGURE IV.

Lateral view in living subject, showing smaller left sinus, which contains slight amount
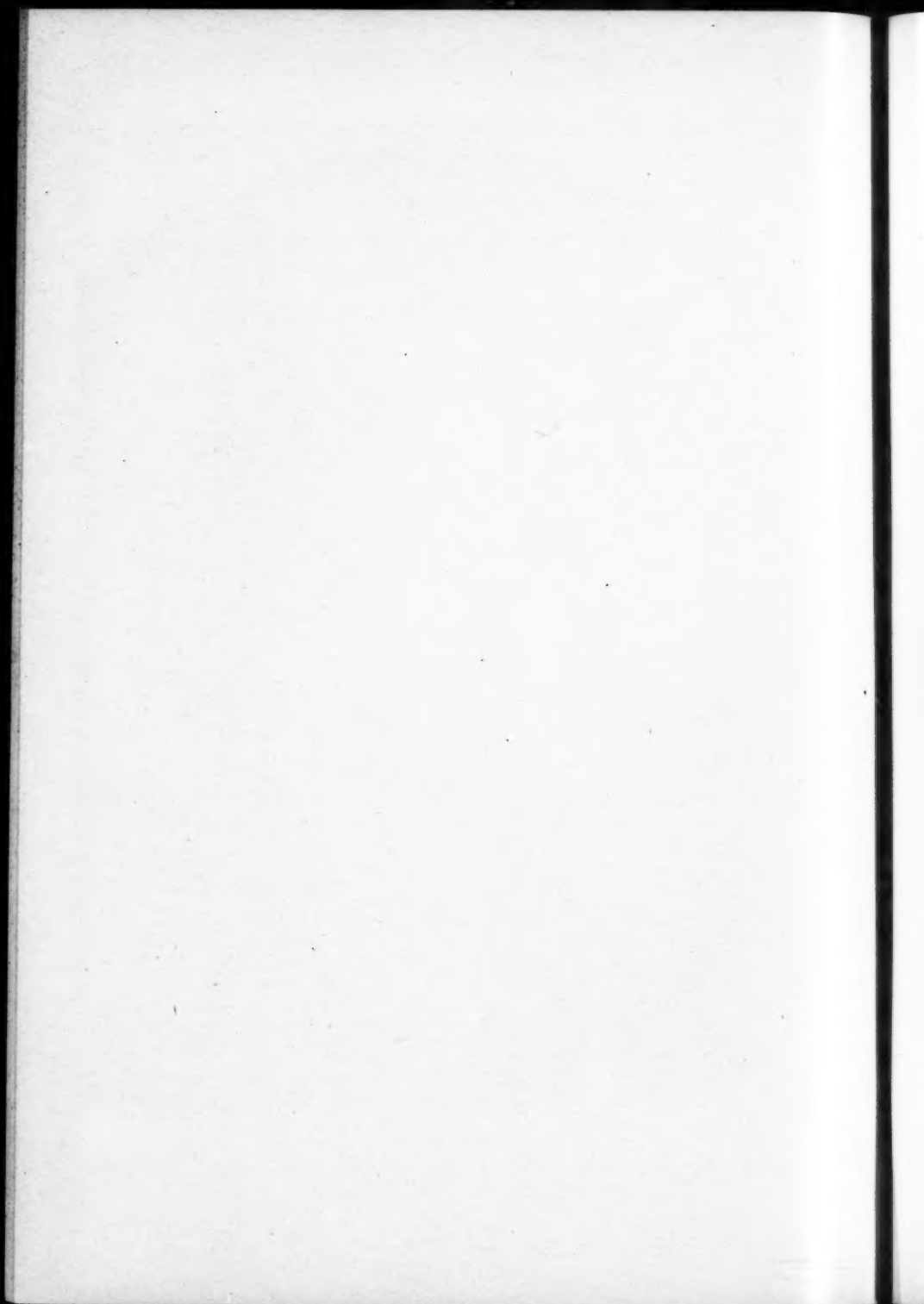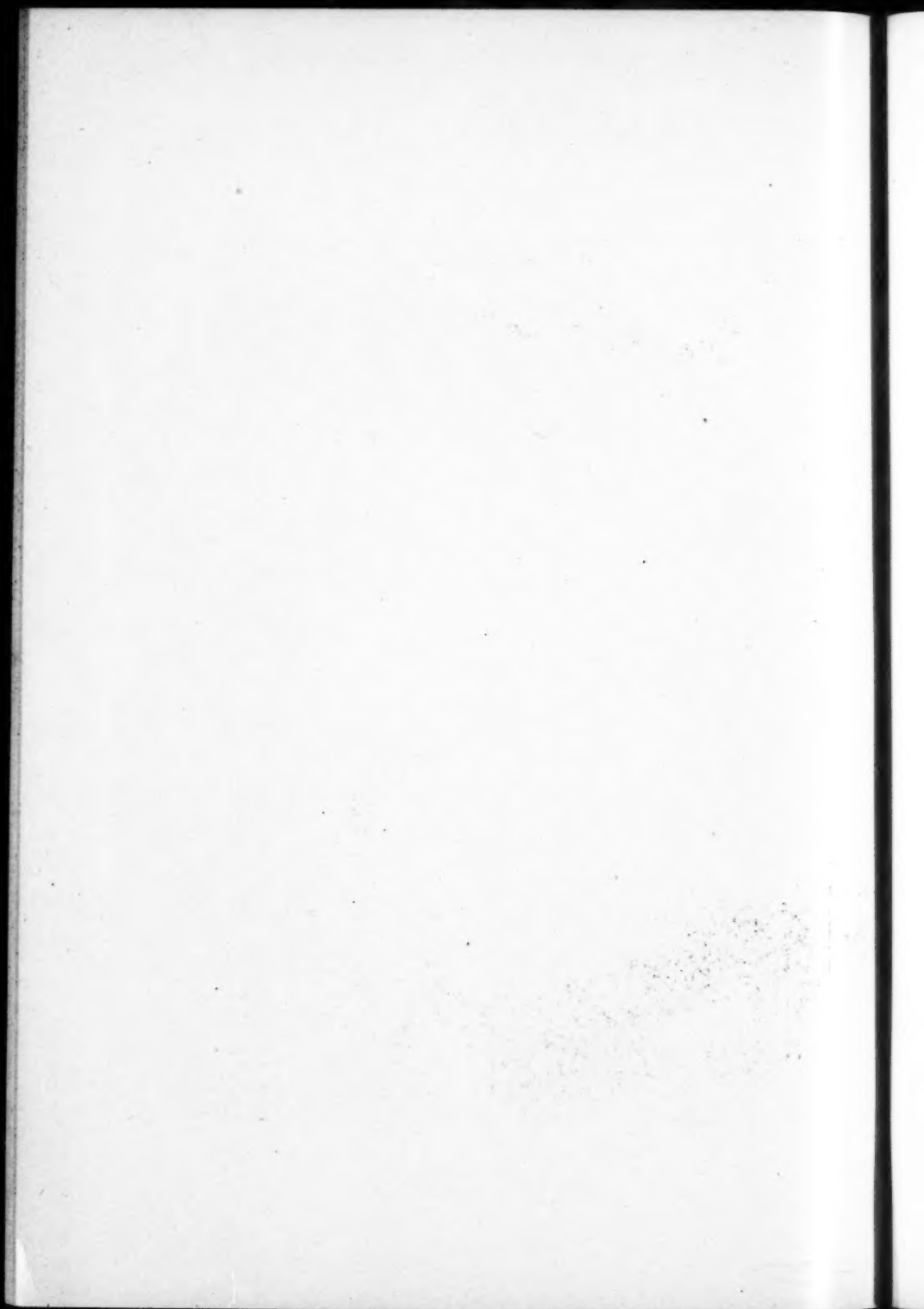of exudate, projected inside of the larger right.

FIGURE V.

Showing posteroanterior view of same patient as Figure IV. The left sinus is smaller than the right and shows slight amount of exudate.

LIII.

PHYSICAL ECONOMICS—WITH REFERENCE TO THE ECONOMIC VALUE OF MAN AND THE MEASUREMENT OF DAMAGES TO ANY PART OR PARTS OF HIS BODY, BY STANDARDS OF MEASUREMENT AND MATHEMATICAL FORMULAS, IN A MANNER EQUITABLE TO ALL CONCERNED.*

BY ERASTUS EUGENE HOLT, A. M., M. D., LL.D.,

PORTLAND, ME.

Any physician who devotes more or less of his time to the treatment of diseases of the ear may be called upon to give expert testimony as to the damages to the ears, from injury or disease, either alone or in connection with damages to other parts of the body. For this reason there should be some standard method by which all those who are called upon to render this service may arrive at definite conclusions which will agree with all the existing conditions of the person who has suffered such damages.

The aim of this paper is to present a standard method of procedure which may be employed by any physician who may be called upon to render such a service. By such a method definite conclusions may be reached, and although the conclusion reached by one physician may differ from that of another, in a given case, nevertheless, these conclusions will have been reached by the same method, and this difference may be readily accounted for by examining the several factors employed and noting the different values given to these factors. Thus every step of the process of each independent observer may be taken up separately, compared with one another, and discussed. Each may state his reasons for determining the value given to each factor in the problem. If an agreement cannot be arrived at in regard to the value given to each factor, a note may be made of

---

*Read before the Forty-fifth Annual Meeting of the American Otological Society. 1912.

this disagreement, and this difference in the values of the factors will indicate precisely why the results differ from one another. Thus all physicians who are called upon to determine damages to any part, or parts, of the body, from injury or disease, will be employing one uniform method corresponding precisely to that by which the value of any physical force is obtained. This method is known as the natural science method. This method was used by the scientists when they devised a way to measure electricity and defined the unit of electromotive force to be a volt, named in honor of Volta, an Italian physicist, which consists of one ampere, named in honor of Ampere, a French physicist, with a resistance of one ohm, named in honor of Ohm, a German physicist. These three elements when used as factors and multiplied together are equal to a watt, named in honor of Watt, the great English inventor. America, the home of some of the greatest inventors, is not represented. If we use the first letter of each of these words as a symbol to represent them as factors in a formula, we have as follows: $V \times A \times O = W$, 746 of which watts equal a horse power. A horse power was designated by Watt, when he invented the steam engine, to be equal to a force which will lift 3,300 pounds one foot in a minute.

With reference to the economic value of man, we find that in the seventeenth century Sir William Petty was the first to attempt to determine the economic value of man from his earning ability. In the latter half of the same century, Adam Smith of Scotland, in that classical work entitled "Nature and Causes of the Wealth of Nations," compasses the whole subject in four sentences, as follows:

"When any expensive machine is erected, the extraordinary work to be performed by it before it is worn out, it must be expected, will replace the capital laid out upon it, with at least the ordinary profits. A man educated at the expense of much labor and time to any of those employments which require extraordinary dexterity and skill may be compared to one of those machines. The work which he learns to perform, it must be expected, over and above the usual wages of common labor, will replace to him the whole expense of his education, with at least the ordinary profits of an equally valuable capital. It must do this, too, in a reasonable time, regard being had to the very un-

certain duration of human life, in the same manner as to the more certain duration of the machine."

Since that time scores of writers have devoted more or less time to the subject. It remained, however, for William Farr, in the nineteenth century, who spent his life in the Registrar-General's office of England, writing on vital statistics and allied subjects, to become an authority on these subjects throughout the whole civilized world. Dr. Farr comprehends this subject in one sentence, as follows:

"The value of any class of lives is determined by valuing first, at birth, or any age, the cost of future maintenance and the value of future earnings."

Ernst Engel of Germany, in a work published in Berlin in 1883 upon this subject, for the extracts from which I am indebted to Professor Wilcox of Cornell University, computed the cost of the production of human beings from household account books. He was not inclined to accept Farr's method, not, however, giving any reason therefor. Engel, after an elaborate consideration of the subject, developed tables of the value of human beings from the point of view of the cost of production. He, however, omitted to reckon one of the chief factors in the production of a human being, namely, the value of the services of the mother. When these services of the mother are added to the cost of production which he obtained without them, they make the expense of producing a human being similar to that obtained by Farr. For economic purposes we must determine the economic value of man from his earning ability, no matter how much he may have cost to produce. The same law of average is a determining factor in this, as in the problem of establishing a life table.

To those who may protest against the determination of the money value of man, Engel quotes from the classical work on anthropometry of Quetelet of France, published in 1885, as follows:

"My investigations and standpoint are so positive that many may shrink from them, some finding them materialistic, others finding in them a desire to extend unduly the limits of the exact sciences and apply mathematics in a region where it does not belong."

Appealing to the advancements made in astronomy in refutation of such ideas and sentiment, Quetelet further says:

"Who will accuse the learned because they have given us in place of the narrow world of the ancients a knowledge of our glorious solar system and have pushed the limits of our starry firmament so far that the mind cannot think of these distances without a religious awe? Surely a knowledge of the wonderful laws that govern the world and have been discovered by learned men makes the power of the divine seem far greater than it did under that conception of the world which a blind superstition would have compelled us to retain."

No. 9, The Evom Table. This word is derived from the first letter of each of the words in the phrase, "economic value of man." It is based upon the method promulgated by Farr, the unit adopted being one dollar a day, or three hundred dollars a year. By adopting this unit, computations may be readily made for a person earning any fractional part of one dollar a day, or three hundred dollars a year, or any number of times one dollar a day, or three hundred dollars a year. The rate of discount upon which the computations are made is three and a half per cent, compound interest, per annum.

The earning ability of a person is a composite quantity made up of certain indispensable elements which may be used as factors in a mathematical formula. These are first what nature gives to man, namely, the structures and functions of the different systems and organs. No one can question the necessity of having these in a fairly healthy state for the normal earning ability of a person. These structures and functions of the different systems and organs of the body may be designated the functional ability of the body, and may be represented by F as a symbol to be used as a factor in the formula.

The structures and functions of the different systems and organs of the body would have no earning ability if they were not trained to perform certain work successfully. This second indispensable element of the earning ability, the training of the mind and body, is given to man by man. It may be termed the technical ability, and be represented by T as a symbol to be used as a factor in the formula.

We now come to the third and last indispensable element of the earning ability, namely, the ability a person has from the training of his mind and body to use them successfully in some vocation and receive a remuneration for such services. This third indispensable element of the earning ability may be des-

ignated the competing ability, and may be represented by C as a symbol to be used as a factor in the formula.

For the earning ability, E may be used as a symbol to represent this composite quantity in the formula, and we have:

(1) $F \times T \times C = E$, the mathematical formula for the normal earning ability of a person, and for determining damages to the body from injury or disease.

In order to have a practical method for determining damages to the ears, from injury or disease, we must have one that can be used for determining damages to the structure and functions of any other system or organ of the body that may have occurred at the same time. A method that could be used only for one set of organs, like the ears or eyes, and could not be used to ascertain damages to other parts of the body, which may have occurred at the same time, would be of little or no value, because one of the parts of such a method would be purely empirical, and, like the chain which is no stronger than its weakest link, it would be no better than its weakest part. It has been necessary, therefore, for this purpose to devise this mathematical formula for the normal earning ability of the entire body, in order that by it we might be able to determine damages to the structures and functions of all the different systems and organs of the entire body, which may occur, either singly, or together, at any one time, by one uniform method, which corresponds to that by which the value of any physical force is obtained. It will be seen that it is necessary to treat this subject on the broad general principle of determining damages to any part, or parts, of the body which may have occurred, either singly, or together, at any one time.

I shall endeavor to make it as clear as possible for the time and space at my disposal.

We found by analysis that E, the earning ability of a person, is a composite quantity, and by the same method we find that its principal factor, F, the functional ability of a person, is also a composite quantity, made up of the different systems and organs of the body. By this analysis, systems and organs have been selected and grouped together into four units, in accordance with their development and associated functions, and each of these four units has also been divided into three parts which include all the indispensable elements of the unit. We thus have four units, each one of which has three parts, making in

all twelve parts which include the structures and functions of
all the indispensable elements of the body which may be dam-
aged from injury or disease.

The four units and each of their subdivisions are represented
by the first letters of the alphabet as symbols to be used as fac-
tors in the formula for determining their remaining functions
when damaged from injury or disease. Letters of the alphabet
have been used only once as symbols, and those which might
lead to confusion in the formulas have not been taken for
symbols.

$$
F = \begin{cases}
a = \begin{cases} \text{Osseous, articular and} \\ \text{muscular systems, con-} \\ \text{sisting of} \dots \dots \dots \end{cases} \begin{cases} \text{h, the bones.} \\ \text{i, the joints.} \\ \text{k, the muscles.} \end{cases} \\[2em]
b = \begin{cases} \text{Circulatory and respira-} \\ \text{tory systems, consisting} \\ \text{of} \dots \dots \dots \dots \end{cases} \begin{cases} \text{m, the vascular system.} \\ \text{n, the blood.} \\ \text{p, the lungs and their ac-} \\ \text{cessory organs.} \end{cases} \\[2em]
d = \begin{cases} \text{Digestive and genitouri-} \\ \text{nary systems, consisting} \\ \text{of} \dots \dots \dots \dots \end{cases} \begin{cases} \text{q, the alimentary canal} \\ \text{and its accessory or-} \\ \text{gans.} \\ \text{r, the skin.} \\ \text{s, the kidneys with the} \\ \text{genital organs.} \end{cases} \\[2em]
g = \begin{cases} \text{Cerebrospinal system,} \\ \text{nerves and organs of} \\ \text{special sense, consisting} \\ \text{of} \dots \dots \dots \dots \end{cases} \begin{cases} \text{u, the brain, its mem-} \\ \text{branes, and its nerves.} \\ \text{v, the spinal cord, its} \\ \text{membranes, and its} \\ \text{nerves.} \\ \text{w, nerves and organs of} \\ \text{special sense.} \end{cases}
\end{cases}
$$

We are thus able to determine the structural and functional
condition of each system or organ, separately or together, in
accordance with the actual conditions of the disability in them.

The factors of a unit multiplied together are equal to the
unit, thus: (2) $h \times i \times k = a$. The units as factors multiplied
together are equal to the functional ability of the body, thus:
(3) $a \times b \times d \times g = F$. The functional ability of the body multi-
plied by the other two factors, T and C, of the earning ability
is equal to E, the earning ability of the person, thus: (4) $F \times T \times C = E$. Thus the values of the twelve different parts, rep-
resenting all the structures and functions of the body, are deter-
mined as readily as the value of one organ, for instance that of
the eye, by one uniform method similar to that which is em-

ployed in the natural sciences to ascertain the value of any physical force.

The next and last requirement for the solution of this problem is a standard of measurement for each of the different systems and organs included in the twelve different factors of the four units representing all the structures and functions of the body.

The various means at our disposal for determining the scientific standard of measurement for normal hearing I shall not consider, for they are the common property of all, and it would take me beyond the scope of my subject to do so. We must, however, always keep in view the law of average in determining it, the same as has been done in determining the scientific standard of measurement for normal sight. Whatever scientific standard of measurement for normal hearing may be adopted, it can be divided into tenths for convenience of record. The full scientific standard of measurement for normal hearing is no more needed for most vocations than the full scientific standard of measurement for sight is needed for most vocations.

The range of sight for the economic standard of measurement established by Magnus of Germany for persons in vocations requiring higher acuity of sight is from 0.15 to 0.75, or a range of six of the ten tenths into which the scientific standard of measurement is divided. For persons in vocations requiring lower acuity of sight the range is from 0.05 to 0.5 of the scientific standard of measurement.

After weighing, measuring, comparing and testing all the requirements of the hearing for the average vocations, the conclusion was reached that a range from 0.1 to 0.7 of the scientific standard of measurement is sufficient for the economic standard of measurement. Table 7 has been constructed upon that basis. By this standard of measurement a person may lose 0.3, according to the scientific standard of measurement, and still have hearing sufficient for economic purposes. Again, if he loses 0.9, according to the scientific standard of measurement, he has 0.1 left, but this is not sufficient for economic purposes and that person would then have a total loss of hearing for economic purposes. By this standard we have a range of 0.6 of the scientific for the economic standard, which is sufficient for all practical purposes.

For convenience we have conformed partly to the terms employed in the Bureau of Pensions of the United States to designate the different degrees of the loss of hearing, on account of their long use and consequent familiarity among medical men. These terms are as follows:

First, slight; second, severe; third, nearly total; and fourth, total loss of hearing.

By examining Table 7, Standard of Measurement for the Loss of Hearing, it will be observed that it corresponds to Table 6, Standard of Measurement for the Loss of Sight, and also to Table 8, Standard of Measurement for the Loss of Smell, for the Loss of Taste, and for the Loss of Feeling.

With the formulas and tables herewith given, we have a scientific and practical method for determining the economic value of man and the measurement of damages to any part, or parts, of his body, from injury or disease, in a manner equitable to all concerned.

It will be noticed in tables from No. 1 to 4, that T, the technical ability of the mathematical formula for the normal earning ability of the body, is omitted, because T, the technical ability, is not injured, but limited in proportion to the damages to F, the functional ability of the body, whose impairment includes these damages. T, the technical ability, after it becomes fixed in life remains one, and for this reason also may be discarded, because multiplying any quantity by one does not change its value.

We thus have the mathematical process of the formula to correspond to the simple terms in arithmetic, namely, F, the functional ability of the person, corresponds to the multiplicand, and C, the competing ability of the person, corresponds to the multiplier, while E, the earning ability of the person, corresponds to the product.

The axiom in arithmetic is that increasing the multiplier increases the product, and diminishing the multiplier diminishes the product; that when the divisor is less than one, the quotient will be greater than the dividend, and that the reverse of a number is one divided by that number.

In Table 1, the coefficient of C, the competing ability, has been increased (by evolution) to have E, the earning ability, increased whenever it is determined, in a given case of injury,

that C, the competing ability, has been damaged to a lesser degree than F, the functional ability.

In Table 2, the coefficient of C, the competing ability, has been diminished (by involution) to have E, the earning ability, decreased whenever it is determined, in a given case of injury, that C, the competing ability, has been damaged to a greater degree than F, the functional ability.

Table 1 represents the reverse process of Table 2, and may be readily understood by the following illustrations: If we produce involution of $1/2$ to the 6th power, we express it thus, $1/2^6$, and it equals $1/2 \times 1/2 \times 1/2 \times 1/2 \times 1/2 \times 1/2 = 1/64$, reduced to a decimal $= 0.015625$. In this example $1/2$ is the multiplicand and $1/2$ is the multiplier. The product is just as much less than the multiplicand as the multiplier is less than one, namely, it is one-half of it every time it is multiplied. This is the reason why raising a fraction by involution, to any given power, diminishes its value. This is what is done in Table 2 to C, the competing ability, in order to have the damage to E, the earning ability, diminished to correspond to the condition of greater damage to C than to F, the functional ability.

In Table 1, the reverse of involution is produced, namely, evolution, and is indicated by the reverse of the number, or one divided by the number, for the index of the exponent, or the root to be sought, thus, $1/64^{1/6}$ or $0.015625^{1/6} = 1/2$ or 0.5. If we did not want to increase the value of the fraction to so high a degree, we would practice evolution a less number of times, as indicated by a smaller number for the denominator of the fractional exponent, or the root to be sought, thus:

$1/64^{1/3}$, or $0.015625^{1/3} = 1/4$, or 0.25.

$1/64^{1/2}$, or $0.015625^{1/2} = 1/8$, or 0.125.

This is what is done in Table 1 to C, the competing ability, in order to have the damage to E, the earning ability, increased to correspond to the condition of lesser degree of damage to C than to F, the functional ability. We have gone into this explanation of the formation of Tables 1 and Tables 2, in order to answer questions that have been asked so many times about them.

In normal conditions the coefficient of F, the functional ability, is one, and as C, the competing ability of a person, is composed of the same elements, its coefficient is one also, hence E, the earning ability, would be one. When a person has sus-

tained damages to his body, the coefficient of F, and of C, becomes less than one, according to the amount of damage sustained. We then have to consider, first, whether the damage to F, the functional ability, causes the same degree of damage to C, the competing ability, or second, whether it causes a less degree of damage to C, or third, whether it causes a greater degree of damage to C, than to F, in the vocation of the person.

The treatment of these conditions in the tables is as follows: First, when it is determined that the damage to F, the functional ability, causes the same degree of damage to C, the competing ability, in the vocation followed by the person, then E, the earning ability, will be the product of these coefficients. (Table 1, Col. 3.)

Second, when it is determined that the damage to F, the functional ability, causes a lesser degree of damage to C, the competing ability, in the vocation followed by the person, then its coefficient must be increased by evolution before it is multiplied by the coefficient of F (Table 1, Cols. 4, 5, 6, 7, 8 and 9), in order to have E, the earning ability, increased to correspond to the lesser degree of damage to C, the competing ability.

Third, when it is determined that the damage to F, the functional ability, causes a greater degree of damage to C, the competing ability, in the vocation followed by the person, then its coefficient must be diminished by involution (Table 2, Cols. 2, 3, 4, 5, 6, 7, 8, 9 and 10), in order to have E, the earning ability, diminished to correspond to the greater degree of damage to C, the competing ability.

In Table 1, E, the earning ability, after damage to F, the functional ability, from 0.01 to 0.80 is given with C, the competing ability, damaged to the same degree as F, and with C damaged to the tenth degree less than F, and in Table 2, with C damaged to the tenth degree more than F, thus giving sixteen different degrees of damage to C, the competing ability, in the two tables. Table 3 and Table 4 are complementary tables to Table 1 and Table 2, giving the loss on one thousand dollars of the economic value of the person for each of these computations. Thus all the computations are made in an accessible form in these tables and will facilitate the work of solving the problem of determining damages to the body from injury or disease. Table 5 explains itself. It may be used as a standard for comparison in damages for other injuries.

We will now give examples illustrating how readily damages to any part, or parts, of the body may be determined by one uniform method by the use of the formulas and the tables.

We will determine the damages (1) to a person forty-three years old, who is a clerk in a department store, for the total loss of hearing in one ear. He is earning two dollars a day, and, therefore, his gross economic value is, in round numbers, $10,000. (No. 9, Evom Table, Col. 2.) The damage to F, the functional ability of the body, according to Table 7, Col. 5, is 0.12. Subtracting this from 1, we have 0.88 for the coefficient of F and C, which introduced into the formula for the normal earning ability of the body, gives: (5) 0.88 F x 0.88 C=E. If we determine that C, the competing ability of this person, is damaged to the sixth degree less than F, the functional ability, we have: (6) 0.88 F x 0.88 C $^1/^7$=E, and we find the value of E, the earning ability, to be 0.8641 (Table 1, Col. 8). If this is the remaining earning ability, we find the loss by subtracting it from 1, and we have 0.1359, the loss on one dollar, or $135.90, the loss on $1,000 (Table 3, Col. 7). As we have just shown that his economic value was $10,000, his economic loss would be ten times $135.90, or $1,359 for the total loss of hearing in one ear. If we add to this the loss in time and expenses incident to the accident, and the loss in suffering in mind and body in consequence of the accident, which was decided to be in this case $336.00, we have a total indemnity of $1,695 for the total loss of hearing in one ear.

We will now determine (2) the damages to a similar clerk for the total loss of hearing in both ears. The damage to F, the functional ability of the body, is twice that for the damage of one ear (Table 7, Col. 5), namely, 0.24. Subtracting this from 1, we have 0.76 for the coefficient of F and C, which introduced into the formula for the normal earning ability of the body, gives: (7) 0.76 F x 0.76 C=E. It is plainly evident that a person with the total loss of hearing in both ears is damaged as much in C, his competing ability, as in F, his functional ability. If, after due consideration of all the conditions and circumstances in this case, we decide that C, his competing ability, is damaged to the same degree as F, his functional ability, we have: (8) 0.76 F x 0.76 C=E. Hence E=0.5776 (Table 1, Col. 3), and the loss is 0.4224 on one dollar, or $422.40 on each $1,000 of the economic value of the person

(Table 3, Col. 2). Multiplying this loss by ten, because his economic value was $10,000, we have $4,224. If we add to this loss, the loss in time and his expenses incident to the accident and the loss in suffering in his mind and body in consequence of the accident, which in this case was decided to be $596, we have the total indemnity of $4,820 for the injury which caused the loss of hearing in both ears. This is $3,125 more indemnity than for the total loss of hearing in one ear.

An indemnity for the loss of any part of the hearing in one ear, or in both ears, as given in Table 7, may be readily worked out in the same way. The question to be determined is the amount of the loss of the hearing in one ear, or in both ears, and the effect this loss has upon C, the competing ability, in the vocation of the person.

We will now show how readily we can determine damages to any part, or parts, of the body, which may have occurred at the same time as the accident which caused the loss of hearing.

We will determine (3) what the indemnity shall be for the total loss of the hearing in one ear, and the total loss of sight of one eye. Both of these losses occur in unit g, factor w, and therefore may be added together (Tables 6 and 7, Col. 5). We find the same to be 0.30. Subtracting this from 1, we have 0.70 for the coefficient of F and C, and we have: (9) 0.70 F x 0.70 C=E. After due consideration of all the conditions and circumstances of C, the competing ability of this person in his vocation as a clerk in the department store, it is decided that C, his competing ability, is damaged to the sixth degree less than F, the functional ability of the body, and we have: (10) 0.70 F x 0.70 $C^1/^7$=E. Hence E, his earning ability, will be 0.6650 (Table 1, Col. 8), and the loss on one dollar is 0.335, or $335 on $1,000 (Table 3, Col. 7). Multiplying this by ten, we have $3,350 as an indemnity for the total loss of hearing in one ear and the total loss of sight of one eye. If we add the loss of his time and expenses and the loss in suffering in his mind and body in consequence of the injury, which was in this case decided to be $324, we have $3,674 as a total indemnity for the total loss of hearing in one ear and the total loss of sight in one eye.

For example (4), the indemnity for the total loss of hearing in both ears and for the total loss of sight in both eyes for a person earning a similar amount would be ascertained as fol-

lows: In Table 6 and Table 7, we find for these four losses 0.60. Subtracting this from 1, we have 0.40 for the coefficient of F and C, the functional and technical abilities, hence: (11) 0.40 F x 0.40 C=E. The question arises at once, what is the condition of C, the competing ability, in this person, totally blind and deaf? It is plainly evident that he has no competing ability, hence the exponent of C, becomes 0, and we express it in the formula thus: (12) 0.40 F x 0.40 C^0=E. Therefore, E=0, because in any computation, when one of the factors becomes 0, the product is 0 also. The loss would be total, or $10,000. If we add the loss in time and expenses before this indemnity is paid and the loss in suffering in mind and body in consequence of this serious loss of both sight and hearing, which in this case was determined to be $2,500, we have $12,500 for the total indemnity for this person, forty-three years old, earning $2.00 per day. If he were earning $3.00 per day, or $900 a year, the indemnity would be $17,500, assuming the expense for loss of time and suffering in mind and body were the same. If he were earning $4.00 per day, or $1,200 per year, the other expenses being the same, the indemnity would be $22,500, and so on. The only other way of determining damages to the body is by empirical methods, using addition and subtraction. This example shows how absurd this would be, for if the other two factors in unit g were normal, which is a conceivable condition, because this person might be totally blind and totally deaf, and the other two factors, u, the brain, and v, the spinal cord, might be normal, and we obtained the loss by subtraction, we would have two factors of this unit left. If the other three units of the body were normal, which is also a conceivable condition, we would have nine other factors, which added to the two of unit g would make eleven. Therefore, by subtraction and addition, we would have 11/12 of F, the functional ability of the body, left when that person was totally blind and deaf.

We will (5) determine the loss to a person earning a similar amount, who in an accident sustains a total loss of hearing in one ear, the total loss of sight in one eye, and the loss of the left hand at the wrist joint. According to Table 5, the loss in unit a is 0.20, and therefore 0.80 is left. According to Table 6 and Table 7, the loss to unit g is 0.30, and therefore 0.70 is left. As these losses occur in two different units their coeffi-

cients must be multiplied together to determine F, the remaining functional ability of the body, and we have: (13) 0.80 a x 0.70 g=F. Hence, F=0.56. If we determine from all the circumstances in this case that C, his competing ability, is damaged to the first degree more than F, his functional ability, we have: (14) 0.56 F x 0.56 C^2=E. Therefore, E=0.1756 (Table 2, Col. 2), and the loss is 0.8244 on one dollar, or $824.40 on $1,000, and ten times that on $10,000, or $8,244. If we add to this loss the other two losses incident to the injury, which were decided to be $1,648, we have $9,892 as the total indemnity for the total loss of hearing in one ear, total loss of sight in one eye and the loss of the left hand at the wrist joint, in a person earning two dollars per day, at the age of forty-three.

For example (6), we assume that a clerk, forty-three years old, in a department store, died from an accident that made the corporation liable for the loss of his life. In the five examples given here to illustrate the application of our method, the damage has been adjusted upon the gross economic value. When a person survives an accident the damage must be adjusted upon that basis, because it costs that person just as much to live after the accident as before it occurred. When a person does not survive an accident but dies, there are no living expenses incurred for him. Hence, the damage must be adjusted upon that basis, which is his net economic value. The net economic value of a person is what he earns less his own personal living expenses (No. 9, Evom Table). In this case the clerk was earning $2.00 per day, or $600 per year, and his personal living expenses were decided to have been $1.00 a day, or $300 per year. Therefore, his net economic value is just half $10,000, his gross economic value, or $5,000 (No. 9, Evom Table, Col. 2). This means that $5,000 put at interest and paid out in the form of an annuity, at 3½ per cent—the same rate by which his economic value is computed—will last just as long as the clerk was expected to live and earn this amount of money over and above his personal expenses, according to the law of average. The expenses incident to the accident and the loss in suffering in mind and body in consequence of the accident were agreed upon as $1,000, which added to $5,000 makes $6,000 as the total indemnity for the corporation to pay for the accident which caused the loss of the life of this clerk,

earning $2.00 per day, or $600 per year, at the age of forty-three.

In 1904, I read a paper before the National Association of United States Pension Examining Surgeons and Members of the Bureau of Pensions of the United States at Atlantic City, in which I referred to the fact that the pensions then given were not only based upon empiricism, but were full of inconsistencies and absurdities, and did not do justice to the soldiers and sailors, and therefore should be discarded for scientific methods which would do away with absurdities and do justice to them. The result was that the pensions were revised, and this revision took effect in ten of the principal pensions in 1905, giving a total increase of $1,968 each year.

To illustrate some of the inequalities and absurdities existing in the Bureau of Pensions, reference may be made to the two standards for disabilities. The standard for minor disabilities is anchylosis of the wrist joint, which is rated 8/18, and a pension of $8.00 per month is given. If any disability is considered to be equal to anchylosis of the wrist, it is rated the same, and a pension for the same amount is given. If a disability is considered to be any fractional part of 8/18, it is rated accordingly; for instance, nearly total deafness in one ear is thought to be three-fourths of the disability of anchylosis of the wrist, and is therefore rated 6/18, and a pension of $6.00 per month is given, while total deafness in one ear is considered to be one and a fourth times as much disability as anchylosis of the wrist, and therefore, is rated 10/18, and a pension of $10 per month is given. If the pension for nearly total loss of hearing in one ear is increased to correspond with the increase in the pension for total disability from $72 to $100 per month, it should now be $8.32 per month, and for total loss of hearing in one ear it should now be $13.88 per month. If we campare these pensions with those which may be obtained by the scientific methods of physical economics, we find, according to Table 7, Col. 5, the standard of measurement for nearly total loss of hearing of one ear of the 1° is 0.08. Subtracting this from 1 we have 0.92, which becomes the coefficient of F and C. The other ear being normal, C, the competing ability of the person, would be damaged in most vocations to the least degree, and we have the formula:
(15) $0.92 \ F \times 0.92 \ C^{1/10} = E$. Hence, $E = 0.9126$ (Table 1,

Col. 9), and the loss would be 0.874, for which a pension of $8.74 should be given. The total loss of hearing in one ear has been worked out in one of the examples already given: (5) 0.88 F x 0.88 C $^{1}/^{7}$=E. Hence, E=0.8641, and the loss is 0.1359, and a pension of $13.59 should be given when the pension for total disability is $100 per month.

The loss of sight in one eye is considered a disability of one and a half times that of anchylosis of the wrist, and therefore is rated 12/18, and a pension of $12 per month is given, while the loss of an eye is considered two and one-eighth times the disability of anchylosis of the wrist, and therefore is rated 17/18, and a pension of $17 per month is given. If the pensions for total loss of sight in one eye, and for the total loss of an eye, are increased to correspond with the increase in the pensions for total disability from $72 to $100 per month, the former should be increased from $12 to $16.67 per month, and the latter from $17 to $23.61 per month. This is about the same amount as is obtained when the total loss of sight in one eye is determined to affect C, the competing ability of a person, to the least degree, in the vocation he follows, as expressed in the formula for the normal earning ability of the body: (16) 0.82 F x 0.82 C $^{1}/^{10}$=E. Hence, E=0.8036 (Table 1, Col. 9), and the loss is 0.1964, and a pension of $19.64 per month should be given for the total loss of sight in one eye. For the total loss of an eye, the damage to C, the competing ability, would be greater, and if in this loss it is determined that C, the competing ability, is damaged to the second degree less than F, the functional ability, in the vocation followed, we have: (17) 0.82 F x 0.82 C $^{1}/^{8}$=E. Hence, E=0.7675 (Table 1, Col. 5), and the loss is 0.2325, for which loss a pension of $23.25 per month should be given for the total loss of an eye.

Minor disabilities are rated from 2/18 to 17/18, and a pension is given in dollars to correspond with the number of the numerator. Although the pension for total disability was increased from $72 per month to $100 per month, the pensions for minor disabilities have not been increased accordingly.

The other standard is the degree of disability equivalent to the loss of a hand, or a foot, and a pension of $24 per month is given. This method does not provide for a pension to be given for $18, $19, $20, $21, $22 or $23 per month, as if there were no disabilities for which pensions could be given for these

amounts. When a disability is considered equivalent to that of the loss of a hand or foot, it is rated the same, and a pension of $24 per month is given. When, however, a soldier or sailor has actually lost a hand at the wrist joint, or a foot at the ankle joint, a pension of $30 per month is given. This difference of $6 per month may have been in recognition of the greater damage to the competing ability of a soldier or sailor when he had actually lost a hand or a foot than when he suffered from a disability equivalent to the loss of a hand or a foot, because when a hand or a foot was lost it could be seen by everybody, whereas the equivalent disability might not be so readily seen. When, however, a hand and a foot were lost, a pension of $36 per month was given. In this double disability there was only an increase of $6 per month. This is altogether insufficient and does a great injustice to any soldier or sailor who has sustained such a serious injury, as may be shown from the scientific methods of physical economics. According to Table 5, the standard of measurement for the loss of a hand or a foot is 0.20. Therefore, when this loss alone is sustained and it is expressed in the formula for the normal earning ability of the body, we have: (18) $0.80 \, F \times 0.80 \, C = E$. If the loss were a foot, and we determined that C, the competing ability of the person, was damaged to the first degree less than F, the functional ability, in the vocation followed, then the same would be expressed in the formula for the normal earning ability of the body as follows: (19) $0.80 \, F \times 0.80 \, C^{1/2} = E$. Hence, $E = 0.7152$ (Table 1, Col. 4). The loss would be 0.2848, and, therefore, a pension of $28.48 per month should be given for the loss of a foot when a pension of $100 per month is given for total disability. If the left hand were lost, in a right-handed person, and we determined that C, the competing ability, was damaged to the same degree as F, the functional ability, then it would be expressed in the formula for the normal earning ability of the body as follows: (20) $0.80 \, F \times 0.80 \, C = E$. Hence, $E = 0.64$ (Table 1, Col. 3), and the loss would be 0.36. Therefore, a pension of $36 a month should be given for the loss of the left hand. If the right hand were lost in a right-handed person, and we determined that C, the competing ability of the person, was damaged to the first degree more than F, the functional ability, in the vocation followed, then it would be expressed in the formula for the normal earning

ability of the body as follows: (21) 0.80 F x 0.80 C^2=E. Hence, E=0.512 (Table 2, Col. 2), and the loss would be 0.4880, and a pension of $48.80 per month should be given for the loss of the right hand. If a hand and a foot were lost we find the sum of these two losses in Table 5 to be 0.40, and subtracting this from 1, we have in the formula for the normal earning ability of the body: (22) 0.60 F x 0.60 C=E. We must then determine how this serious loss affects the person in the vocation followed. It is self-evident that the loss of a hand and a foot at the same time would damage C, the competing ability of a person, more than the loss of either one alone. We then determined that the damage to C, the competing ability, was equal to the first degree less than to F, the functional ability, when a foot was lost at the ankle joint, that the former was damaged to the same degree when the left hand was lost, and that the former was damaged to the first degree more than the latter when the right hand was lost. It would be conservative then to decide that when a foot and left hand were lost, C, the competing ability, would be damaged to the same degree as F, the functional ability, and we have: (23) 0.60 F x 0.60 C=E. Hence, E=0.36 (Table 1, Col. 3), and the loss would be 0.64, for which a pension of $64 per month should be given for the loss of a foot and the left hand. If the right hand and a foot were lost it would be conservative to determine that C, the competing ability, was damaged to the first degree more than F, the functional ability, because when the right hand alone was lost, we determined that C, the competing ability, was damaged to the first degree more than F, the functional ability, and we have: (24) 0.60 F x 0.60 C^2=E. Hence, E= 0.216 (Table 2, Col. 2), and the loss would be 0.784, for which a pension of $78.40 should be given for the loss of a foot and the right hand.

In the revision of the pensions brought about by my paper the pension for the loss of a hand and a foot was increased from $36 to $60 per month. That C, the competing ability, is still not duly recognized in the Bureau of Pensions is very evident from the cases just cited of the loss of a foot, a left hand, or a right hand, or when a foot is lost with the right or left hand. The pension for the loss of both hands was established by a vote of Congress to be $100 per month, whereas the pension for total disability, like that of the loss of both eyes,

was $72 per month. The pension for total loss of hearing in both ears was $30 per month. This was increased in the revision of the pensions to $40 per month. The latter is very nearly the same as is obtained in equation (8), $0.76\ F \times 0.76\ C = E$, when $E = 0.5776$ (Table 1, Col. 3), and the damage is 0.4224, or $42.24 per month when the pension for total disability is $100 per month.

These examples might be multiplied indefinitely, but a sufficient number and variety have been given to show how damages to any part, or parts, of the body may be obtained by the formulas and the scientific and economic standards of measurement.

Those who have considered this subject but little often express distrust of any method which employs a mathematical formula that determines E, the earning ability of a person, and his economic value therefrom, in dollars and cents. They say there is so much uncertainty about C, the competing ability of persons, that they do not see how it can be determined with any degree of reliability. When, however, they admit that life itself is the most uncertain thing in this world, and from its existence the law of average is deduced, upon which the life table is constructed, and life insurance, the largest business of the world, is projected and carried on successfully, they become convinced that it is feasible and practical, because they then realize that the principal factor in determining E, the earning ability of a person, and his economic value, depends upon the same law of average as that by which the life table itself is constructed.

There may be a difference of opinion as to the standards of measurement for determining the loss to F, the functional ability of the body, in any given case of damage. The tables here given may not be the final determination in these problems, but they are the best we have to offer after studying the subject for years, and testing them with all the conditions and circumstances connected with such damages, and the results obtained by empirical methods in courts of law and in the Bureau of Pensions of the United States. At first sight the amount given in the tables for the loss specified would seem to be too low, but it must be remembered that the standard of measurement for the functional loss of any part, or parts, of the body must be so low that the loss to F, the functional ability

of the body, would be equal to this amount to a person in any vocation.   In determining these standards of measurement, for the partial and total loss of hearing, this fact, that they must be made so low as to meet this loss equitably in persons of every vocation, has been kept constantly in view.   We feel sure then that when a person has sustained a total or partial loss of hearing in one ear or both ears, and the damage to F, the functional ability of the body, has been adjusted by this table, the loss to E, the earning ability, can always be equitably determined in every case by considering how this damage affects C, the competing ability, in the vocation the person follows.   Hence, we are forced to the conclusion that the standard of measurement determined upon in Table 7 meets and satisfies every condition connected with the partial or complete loss of hearing in one or both ears for determining damages to E, the earning ability of a person, in a manner equitable to all concerned.

TABLE I

(1)	(2)		(3)	(4)	(5)	(6)	(7)	(8)	(9)
Loss in F	Computations made when C, the competing ability, is damaged to the same degree as F, the functional ability, and when C, the competing ability, is damaged to six different degrees less than F, the functional ability								
	F	C	$E=$	$C^{1.2}E=$	$C^{1.3}E=$	$C^{1.4}E=$	$C^{1.5}E=$	$C^{1.7}E=$	$C^{1.10}E=$
0.01	.99	.99	$=0.9801$	0.985	0.9865	0.9875	0.988	0.9884	0.989
0.02	.98	.98	$=0.9604$	0.9702	0.9731	0.9751	0.976	0.9770	0.9780
0.03	.97	.97	$=0.9409$	0.9554	0.9593	0.9622	0.9641	0.9651	0.9670
0.04	.96	.96	$=0.9216$	0.9408	0.9465	0.9504	0.9523	0.9542	0.9561
0.05	.95	.95	$=0.9025$	0.9262	0.9388	0.9376	0.9405	0.9424	0.9452
0.06	.94	.94	$=0.8836$	0.9118	0.9202	0.9259	0.9277	0.9315	0.9343
0.07	.93	.93	$=0.8649$	0.8965	0.9067	0.9132	0.9151	0.9197	0.9234
0.08	.92	.92	$=0.8464$	0.8822	0.8942	0.9006	0.9043	0.9089	0.9126
0.09	.91	.91	$=0.8281$	0.8681	0.8898	0.8881	0.8927	0.8972	0.9001
0.10	.90	.90	$=0.81$	0.8541	0.8685	0.8766	0.8811	0.8865	0.8901
0.11	.89	.89	$=0.7921$	0.8392	0.8552	0.8641	0.8695	0.8748	0.8784
0.12	.88	.88	$=0.7744$	0.8254	0.8430	0.8527	0.8580	0.8641	0.8685
0.13	.87	.87	$=0.7569$	0.8108	0.8308	0.8404	0.8456	0.8526	0.8578
0.14	.86	.86	$=0.7396$	0.7972	0.8187	0.8290	0.8342	0.8419	0.8571
0.15	.85	.85	$=0.7225$	0.7828	0.8058	0.8160	0.8228	0.8304	0.8347
0.16	.84	.84	$=0.7056$	0.7694	0.7926	0.8047	0.8114	0.8190	0.8257
0.17	.83	.83	$=0.6889$	0.7561	0.7802	0.7885	0.7992	0.8075	0.8142
0.18	.82	.82	$=0.6724$	0.7429	0.7675	0.7806	0.7880	0.7970	0.8036
0.19	.81	.81	$=0.6561$	0.729	0.7549	0.7686	0.7759	0.7857	0.7929
0.20	.80	.80	$=0.64$	0.7152	0.7424	0.7568	0.7648	0.7752	0.7824
0.21	.79	.79	$=0.6241$	0.6915	0.7299	0.7449	0.7528	0.7639	0.7710
0.22	.78	.78	$=0.6084$	0.6887	0.7183	0.7332	0.7417	0.7527	0.7605
0.23	.77	.77	$=0.5929$	0.6752	0.7060	0.7214	0.7299	0.7415	0.7499
0.24	.76	.76	$=0.5776$	0.6627	0.6938	0.7098	0.7189	0.7303	0.7394
0.25	.75	.75	$=0.5625$	0.6495	0.6817	0.6982	0.7072	0.7192	0.7282
0.26	.74	.74	$=0.5476$	0.6371	0.6697	0.6867	0.6963	0.7089	0.7178
0.27	.73	.73	$=0.5329$	0.6241	0.6577	0.6752	0.6847	0.6978	0.7073
0.28	.72	.72	$=0.5184$	0.6112	0.6458	0.6638	0.6739	0.6868	0.6969
0.29	.71	.71	$=0.5041$	0.5985	0.6340	0.6517	0.6624	0.6759	0.6858
0.30	.70	.70	$=0.49$	0.5859	0.6223	0.6405	0.6517	0.6650	0.6755
0.31	.69	.69	$=0.4761$	0.5738	0.6099	0.6285	0.6403	0.6541	0.6679
0.32	.68	.68	$=0.4624$	0.561	0.5984	0.6174	0.6296	0.6432	0.6541
0.33	.67	.67	$=0.4489$	0.5487	0.5862	0.6056	0.6184	0.6324	0.6432
0.34	.66	.66	$=0.4356$	0.5365	0.5748	0.5946	0.6078	0.6217	0.6329
0.35	.65	.65	$=0.4225$	0.5239	0.5629	0.5830	0.5967	0.6110	0.6220
0.36	.64	.64	$=0.4096$	0.512	0.5516	0.5721	0.5856	0.6003	0.6118
0.37	.63	.63	$=0.3969$	0.4995	0.5399	0.5607	0.5745	0.5896	0.6010
0.38	.62	.62	$=0.3844$	0.4879	0.5288	0.5499	0.5635	0.5790	0.5908
0.39	.61	.61	$=0.3721$	0.4758	0.5172	0.5386	0.5526	0.5685	0.5801
0.40	.60	.60	$=0.36$	0.4644	0.5058	0.5280	0.5418	0.5580	0.5700
0.41	.59	.59	$=0.3481$	0.4525	0.4944	0.5174	0.531	0.5475	0.5593
0.42	.58	.58	$=0.3364$	0.4413	0.4837	0.5075	0.5202	0.5370	0.5492
0.43	.57	.57	$=0.3249$	0.4297	0.4725	0.4959	0.5090	0.5261	0.5386
0.44	.56	.56	$=0.3136$	0.4188	0.4614	0.4884	0.4984	0.5157	0.5286
0.45	.55	.55	$=0.3025$	0.4075	0.4504	0.4735	0.4878	0.5049	0.5181
0.46	.54	.54	$=0.2916$	0.3969	0.4395	0.4627	0.4773	0.4946	0.5076
0.47	.53	.53	$=0.2809$	0.3858	0.4287	0.4520	0.4664	0.4838	0.4971
0.48	.52	.52	$=0.2704$	0.3749	0.4180	0.4414	0.4560	0.4737	0.4872
0.49	.51	.51	$=0.2601$	0.3641	0.4080	0.4309	0.4447	0.4630	0.4763
0.50	.50	.50	$=0.25$	0.3535	0.3985	0.4205	0.4350	0.4530	0.4665
0.51	.49	.49	$=0.2401$	0.3430	0.3871	0.4096	0.4243	0.4424	0.4561

TABLE I—Continued

(1)	(2)		(3)	(4)	(5)	(6)	(7)	(8)	(9)
Loss in F			Computations made when C, the competing ability, is damaged to the same degree as F, the functional ability, and when C, the competing ability, is damaged to six different degrees less than F, the functional ability						
	F	C	E=	$C^{1.2}$ E=	$C^{1.3}$ E=	$C^{1.4}$ E=	$C^{1.5}$ E=	$C^{1.7}$ E=	$C^{1.10}$ E=
0.52	.48	.48	= 0.2304	0.3326	0.3758	0.3993	0.4142	0.4324	0.4459
0.53	.47	.47	= 0.2209	0.3219	0.3651	0.3886	0.4037	0.4220	0.4356
0.54	.46	.46	= 0.2116	0.3118	0.3551	0.3785	0.3937	0.4117	0.4255
0.55	.45	.45	= 0.2025	0.3015	0.3447	0.3681	0.3834	0.4014	0.4153
0.56	.44	.44	= 0.1936	0.2917	0.3348	0.3581	0.3735	0.3911	0.4052
0.57	.43	.43	= 0.1849	0.2795	0.3225	0.3478	0.3633	0.3809	0.3951
0.58	.42	.42	= 0.1764	0.2721	0.3145	0.3381	0.3532	0.3708	0.3851
0.59	.41	.41	= 0.1681	0.2624	0.3046	0.3321	0.3431	0.3595	0.3747
0.60	.40	.40	= 0.16	0.2528	0.2948	0.3180	0.3332	0.3504	0.3648
0.61	.39	.39	= 0.1521	0.2433	0.2847	0.3081	0.3229	0.3408	0.3549
0.62	.38	.38	= 0.1444	0.2340	0.2751	0.2983	0.3131	0.3309	0.3450
0.63	.37	.37	= 0.1369	0.2249	0.2652	0.2886	0.3030	0.3207	0.3348
0.64	.36	.36	= 0.1296	0.216	0.2559	0.2791	0.2934	0.3110	0.3250
0.65	.35	.35	= 0.1225	0.2068	0.2481	0.2709	0.2835	0.3010	0.3150
0.66	.34	.34	= 0.1156	0.1982	0.2373	0.2597	0.2740	0.2913	0.3053
0.67	.33	.33	= 0.1089	0.1894	0.2280	0.2501	0.2643	0.2814	0.2953
0.68	.32	.32	= 0.1024	0.1811	0.2188	0.2406	0.2547	0.2720	0.2854
0.69	.31	.31	= 0.0961	0.1726	0.2095	0.2312	0.2452	0.2622	0.2755
0.70	.30	.30	= 0.09	0.1644	0.2007	0.222	0.2358	0.2526	0.2658
0.71	.29	.29	= 0.0841	0.1560	0.1902	0.2125	0.2262	0.2430	0.2560
0.72	.28	.28	= 0.0784	0.1481	0.1831	0.2035	0.2170	0.2335	0.2464
0.73	.27	.27	= 0.0729	0.1401	0.1744	0.1944	0.2076	0.2238	0.2367
0.74	.26	.26	= 0.0676	0.1326	0.1658	0.1856	0.1986	0.2145	0.2272
0.75	.25	.25	= 0.0625	0.1250	0.1572	0.1767	0.1895	0.2050	0.2175
0.76	.24	.24	= 0.0576	0.1176	0.1490	0.1680	0.1804	0.1958	0.2080
0.77	.23	.23	= 0.0529	0.1101	0.1407	0.1591	0.1713	0.1865	0.1984
0.78	.22	.22	= 0.0484	0.1031	0.1328	0.1507	0.1625	0.1773	0.1889
0.79	.21	.21	= 0.0441	0.0961	0.1247	0.1421	0.1537	0.168	0.1795
0.80	.20	.20	= 0.04	0.0894	0.1170	0.1338	0.1450	0.1588	0.1702

TABLE 2

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Loss in F	Computations made when C, the competing ability, is damaged from 2 to 10 degrees more than F, the functional ability								
	$C^2 E=$	$C^3 E=$	$C^4 E=$	$C^5 E=$	$C^6 E=$	$C^7 h=$	$C^8 E=$	$C^9 E=$	$C^{10} E=$
0.01	0.9702	0.9604	0.9507	0.9411	0.9316	0.9222	0.9129	0.9039	0.8948
0.02	0.9415	0.9222	0.9037	0.8856	0.8678	0.8504	0.8333	0.8166	0.8002
0.03	0.9126	0.8852	0.8586	0.8328	0.8078	0.7835	0.7599	0.7371	0.7149
0.04	0.8847	0.8493	0.8153	0.7826	0.7512	0.7211	0.6922	0.6645	0.6379
0.05	0.8573	0.8144	0.7736	0.7349	0.6981	0.6631	0.6299	0.5984	0.5684
0.06	0.8305	0.7806	0.7337	0.6896	0.6482	0.6093	0.5727	0.5383	0.5060
0.07	0.8043	0.7479	0.6955	0.6468	0.6015	0.5593	0.5251	0.4883	0.4541
0.08	0.7786	0.7163	0.6589	0.6061	0.5515	0.5073	0.4667	0.4293	0.3949
0.09	0.7535	0.6856	0.6238	0.5676	0.5165	0.4700	0.4277	0.3692	0.3359
0.10	0.729	0.6561	0.5904	0.5313	0.4781	0.4302	0.3871	0.3483	0.3133
0.11	0.7049	0.6273	0.5582	0.4967	0.4420	0.3933	0.3500	0.3115	0.2772
0.12	0.6814	0.5996	0.5276	0.4642	0.4084	0.3593	0.3161	0.2781	0.2447
0.13	0.6583	0.5727	0.4982	0.4334	0.3770	0.3279	0.2852	0.2481	0.2158
0.14	0.6360	0.5469	0.4703	0.4044	0.3477	0.293	0.2519	0.2166	0.1862
0.15	0.6141	0.5219	0.4436	0.377	0.3204	0.2723	0.2314	0.1966	0.1671
0.16	0.5927	0.4978	0.4181	0.3512	0.295	0.2478	0.2081	0.1748	0.1467
0.17	0.5717	0.4745	0.3938	0.3268	0.2712	0.225	0.1887	0.1566	0.1299
0.18	0.5513	0.4520	0.3706	0.3038	0.2491	0.2042	0.1674	0.1372	0.1125
0.19	0.5314	0.4304	0.3486	0.2823	0.2286	0.1851	0.1499	0.1214	0.0983
0.20	0.512	0.4096	0.3276	0.2620	0.2096	0.1676	0.134	0.1072	0.0853
0.21	0.4930	0.3894	0.3076	0.2430	0.1919	0.1516	0.1197	0.0945	0.0746
0.22	0.4745	0.3701	0.2886	0.2251	0.1755	0.1368	0.1067	0.0832	0.0648
0.23	0.4565	0.3515	0.2706	0.2083	0.1603	0.1234	0.095	0.0731	0.0562
0.24	0.4389	0.3335	0.2534	0.1925	0.1463	0.1111	0.0844	0.0641	0.0487
0.25	0.4218	0.3163	0.2372	0.1779	0.1334	0.1000	0.075	0.0562	0.0421
0.26	0.4052	0.2998	0.2218	0.1641	0.1214	0.0898	0.0664	0.0491	0.0363
0.27	0.3890	0.2839	0.2072	0.1512	0.1103	0.0805	0.0587	0.0428	0.0312
0.28	0.3732	0.2687	0.1934	0.1392	0.1002	0.0721	0.0519	0.0373	0.0268
0.29	0.3579	0.2541	0.1804	0.1280	0.0908	0.0644	0.0457	0.0324	0.023
0.30	0.343	0.2401	0.168	0.1176	0.0823	0.0576	0.0403	0.0282	0.0197
0.31	0.3285	0.2266	0.1563	0.1078	0.0743	0.0512	0.0353	0.0243	0.0167
0 32	0.3144	0.2137	0.1453	0.0988	0.0671	0.0456	0.031	0.0210	0.0142
0.33	0.3007	0.2014	0.1349	0.0903	0.0605	0.0405	0.0271	0.0181	0.0121
0.34	0.2874	0.1896	0.1251	0.0825	0.0544	0.0359	0.0236	0.0155	0.0102
0.35	0.2746	0.1784	0.1159	0.0753	0.0489	0.0317	0.0206	0.0133	0.0086
0.36	0.2621	0.1679	0.1073	0.0686	0.0439	0.028	0.0179	0.0114	0.0072
0.37	0.2500	0.1575	0.0992	0.0624	0.0393	0.0247	0.0155	0.0097	0.0061
0 38	0.2383	0.1477	0.0915	0.0567	0.0351	0.0217	0.0134	0.0083	0.0051
0.39	0.2269	0.1384	0.0844	0.0514	0.0313	0.019	0.0115	0.007	0.0042
0.40	0.216	0.1296	0.0777	0.0466	0.0279	0.0167	0.01	0.006	0.0036
0.41	0.2053	0.1211	0.0714	0.0421	0.0248	0.0146	0.0086	0.005	0.0029
0.42	0.1951	0.1131	0.0655	0.0379	0.0219	0.0127	0.0073	0.0042	0.0024
0.43	0.1851	0.1055	0.0601	0.0342	0.0194	0.011	0.0062	0.0035	0.0019
0.44	0.1756	0.0983	0.055	0.0308	0.0172	0.0096	0.0053	0.0029	0.0016
0.45	0.1663	0.0914	0.0502	0.0275	0.0151	0.0083	0.0045	0.0026	0.0014
0.46	0.1574	0.0849	0.0458	0.0247	0.0133	0.0071	0.0038	0.002	0.001
0.47	0.1488	0.0788	0.0417	0.0221	0.0117	0.0062	0.0032	0.0016	0.0008
0.48	0.1406	0.0731	0.038	0.0197	0.0102	0.0053	0.0027	0.0014	0.0007
0.49	0.1326	0.0676	0.0344	0.0175	0.0089	0.0045	0.0022	0.0011	0.0005
0.50	0.1250	0.0625	0.0312	0.0156	0.0078	0.0039	0.0019	0.0009	0.0004
0.51	0.1176	0.0576	0.0282	0.0138	0.0067	0.0032	0.0015	0.0007	0.0003
0.52	0.1105	0.0530	0.0254	0.0121	0.0058	0.0027	0.0012	0.0005	0.0002
0.53	0.1038	0.0487	0.0228	0.0107	0.0050	0.0023	0.0010	0.0004	0.0001

TABLE 2—Continued

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Loss in F	Computations made when C, the competing ability, is damaged from 2 to 10 degrees more than F, the functional ability								
	$C^2E=$	$C^3E=$	$C^4E=$	$C^5E=$	$C^6E=$	$C^7E=$	$C^8E=$	$C^9E=$	$C^{10}E=$
0.54	0.0973	0.0447	0.0205	0.0094	0.0043	0.0019	0.0008	0.0003	0.0001
0.55	0.0911	0.0409	0.0184	0.0082	0.0036	0.0016	0.0007	0.0003	0.0001
0.56	0.0851	0.0374	0.0164	0.0072	0.0031	0.0013	0.0005	0.0002	0.0000
0.57	0.0795	0.0341	0.0146	0.0062	0.0026	0.0011	0.0004	0.0001	
0.58	0.0740	0.0310	0.013	0.0054	0.0022	0.0009	0.0003	0.0001	
0.59	0.0689	0.0282	0.0115	0.0047	0.0019	0.0007	0.0002	0.0000	
0.60	0.064	0.0256	0.0102	0.004	0.0016	0.0006	0.0002		
0.61	0.0593	0.0231	0.009	0.0035	0.0013	0.0005	0.0001		
0.62	0.0548	0.0208	0.0079	0.0030	0.0011	0.0004	0.0001		
0.63	0.0506	0.0187	0.0069	0.0025	0.0009	0.0003	0.0001		
0.64	0.0466	0.0167	0.006	0.0021	0.0007	0.0002	0.0000		
0.65	0.0428	0.0149	0.0052	0.0018	0.0006	0.0001			
0.66	0.0393	0.0133	0.0045	0.0015	0.0005	0.0001			
0.67	0.0359	0.0118	0.0038	0.0012	0.0003	0.0000			
0.68	0.0327	0.0104	0.0033	0.0010	0.0003				
0.69	0.0297	0.0092	0.0028	0.0008	0.0002				
0.70	0.027	0.0081	0.0024	0.0007	0.0002				
0.71	0.0243	0.007	0.002	0.0005	0.0001				
0.72	0.0219	0.0061	0.0017	0.0004	0.0001				
0.73	0.0196	0.0052	0.0014	0.0003	0.0000				
0.74	0.0175	0.0045	0.0011	0.0002					
0.75	0.0156	0.0039	0.0009	0.0002					
0.76	0.0138	0.0032	0.0007	0.0001					
0.77	0.0121	0.0027	0.0005	0.0001					
0.78	0.0106	0.0023	0.0005	0.0001					
0.79	0.0092	0.0019	0.0003	0.0000					
0.80	0.008	0.0016	0.0003						

TABLE 3

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Loss in F	Loss on $1,000, when C, the competing ability, is damaged to the same degree as F, the functional ability, and when C, the competing ability, is damaged to six different degrees less than F, the functional ability						
	C^1	$C^{1.2}$	$C^{1.3}$	$C^{1.4}$	$C^{1.5}$	$C^{1.7}$	$C^{1.10}$
0.01	$ 19.90	$ 15.00	$ 13.50	$ 12.50	$ 12.00	$ 11.60	$ 11.00
0.02	39.60	29.80	26.90	24.90	24.00	23.00	22.00
0.03	59.10	44.60	40.70	37.80	35.90	34.90	33.00
0.04	70.40	59.20	53.50	49.60	47.70	45.80	43.90
0.05	97.50	73.80	66.20	62.40	59.50	57.60	54.80
0.06	116.40	88.20	79.80	74.10	72.30	68.50	65.70
0.07	135.10	103.50	93.30	86.80	84.90	80.30	76.60
0.08	153.60	117.80	105.80	99.40	95.70	91.10	87.40
0.09	171.90	131.90	110.20	111.90	107.30	102.80	99.90
0.10	190.00	145.90	131.50	123.40	118.90	113.50	109.90
0.11	207.90	160.80	144.80	135.90	130.50	125.50	121.60
0.12	225.60	174.60	157.00	147.30	142.00	135.90	131.50
0.13	243.10	189.20	169.20	159.60	154.40	147.40	142.20
0.14	260.40	202.80	181.30	171.00	165.80	158.10	152.90
0.15	277.50	217.20	194.20	184.00	177.20	169.60	165.30
0.16	294.40	230.60	207.40	195.30	188.60	181.00	174.30
0.17	311.10	243.90	219.80	211.50	200.80	192.50	185.80
0.18	327.60	257.10	232.50	219.40	212.00	203.00	196.40
0.19	343.90	271.00	245.10	231.40	224.10	214.30	207.10
0.20	360.00	284.80	257.60	243.20	235.20	224.80	217.60
0.21	375.90	308.50	270.10	255.10	247.20	236.10	229.00
0.22	391.60	311.30	281.70	266.80	258.30	247.30	239.50
0.23	407.10	324.80	294.00	278.60	270.10	258.50	250.10
0.24	422.40	347.30	306.20	290.20	281.10	269.70	260.60
0.25	437.50	350.50	318.30	301.80	292.80	280.80	271.80
0.26	452.40	362.90	330.30	312.30	303.70	291.10	282.20
0.27	467.10	375.90	342.30	324.80	315.30	302.20	292.70
0.28	481.60	388.80	354.20	336.20	326.10	313.20	303.10
0.29	495.90	401.50	366.00	348.30	337.60	324.10	313.50
0.30	510.00	414.10	377.70	359.50	348.30	335.00	324.50
0.31	523.90	426.70	390.10	371.50	359.70	345.90	334.90
0.32	537.60	439.00	401.60	382.60	370.40	356.80	345.90
0.33	551.10	451.30	413.80	394.40	381.60	367.60	356.80
0.34	564.40	463.50	425.20	405.40	392.20	378.30	367.10
0.35	577.50	476.10	437.10	417.00	403.30	389.00	378.00
0.36	590.40	488.00	448.40	427.90	414.40	397.70	388.20
0.37	603.10	500.50	460.10	439.30	425.50	410.40	399.00
0.38	615.60	512.10	471.20	450.10	436.50	421.00	409.20
0.39	627.90	524.20	482.80	461.40	447.40	431.50	419.90
0.40	640.00	535.60	494.20	472.00	458.20	442.00	430.00
0.41	651.90	547.50	505.60	482.60	469.00	452.50	440.70
0.42	663.60	558.70	516.30	492.50	478.80	463.00	450.80
0.43	675.10	570.30	527.50	504.10	491.00	473.90	461.40
0.44	686.40	581.20	538.60	511.60	501.60	484.30	471.40
0.45	697.50	592.50	549.60	526.50	512.20	495.10	481.90
0.46	708.40	603.10	560.50	537.30	522.70	505.40	492.40
0.47	719.10	614.20	571.30	548.00	533.60	516.20	502.90
0.48	729.60	625.10	582.00	558.00	544.00	526.30	512.80
0.49	739.90	635.90	592.00	569.10	555.30	537.00	523.20
0.50	750.00	646.50	601.50	579.50	565.00	547.00	533.50
0.51	759.60	657.00	612.90	590.40	575.70	557.60	543.90

TABLE 3—Continued

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Loss in F	Loss on $1,000, when C, the competing ability, is damaged to the same degree as F, the functional ability, and when C, the competing ability, is damaged to six different degrees less than F, the functional ability						
	C^1	$C^{1.2}$	$C^{1.3}$	$C^{1.4}$	$C^{1.5}$	$C^{1.7}$	$C^{1.10}$
0.52	769.60	667.40	624.20	600.70	585.80	567.60	554.10
0.53	779.10	678.10	634.90	611.40	596.30	578.00	564.40
0.54	788.40	688.20	644.90	621.50	606.40	588.30	574.50
0.55	795.50	698.50	655.30	631.90	616.60	598.60	584.70
0.56	806.40	708.30	665.20	641.90	626.50	608.90	594.80
0.57	815.10	720.50	677.50	652.20	637.70	619.10	604.90
0.58	823.50	727.90	685.50	661.90	646.80	629.20	614.90
0.59	831.90	737.60	695.40	667.90	656.90	640.90	625.30
0.60	840.00	747.20	705.20	682.00	666.80	649.20	635.20
0.61	847.10	756.70	715.30	691.90	677.10	659.20	645.10
0.62	855.60	766.00	724.90	701.70	686.90	669.10	655.00
0.63	863.10	775.10	734.80	711.40	697.00	679.30	665.20
0.64	870.40	784.00	744.10	720.90	706.60	689.00	675.00
0.65	877.50	793.20	751.90	729.10	716.50	699.00	685.00
0.66	884.40	801.80	762.70	740.30	726.00	708.70	694.70
0.67	891.10	810.60	772.00	749.90	735.70	718.60	704.70
0.68	897.60	818.90	781.20	759.40	745.30	728.00	714.60
0.69	903.90	827.40	790.50	768.80	754.80	737.80	724.50
0.70	910.00	835.60	799.30	778.00	764.20	747.40	734.20
0.71	915.90	844.00	809.80	787.50	773.80	757.00	744.00
0.72	962.60	851.90	816.90	796.50	783.00	766.50	753.60
0.73	927.10	858.90	825.60	805.60	792.40	776.20	763.30
0.74	932.40	867.40	834.20	814.40	801.40	785.50	772.80
0.75	937.50	875.00	842.80	823.30	810.50	795.00	782.50
0.76	942.40	882.40	851.00	832.00	819.60	804.20	792.00
0.77	947.10	889.90	859.30	840.90	828.70	813.50	801.60
0.78	951.50	896.90	867.20	849.30	837.50	822.70	811.10
0.79	955.10	903.90	875.30	857.90	846.30	832.00	820.50
0.80	960.00	910.60	883.00	866.20	855.00	841.20	829.80

TABLE 4

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Loss in F	Loss on $1,000 when C, the competing ability, is damaged from 2 to 10 degrees more than F, the functional ability								
	C^2	C^3	C^4	C^5	C^6	C^7	C^8	C^9	C^{10}
0.01	$ 29.80	$ 39.60	$ 49.30	$ 58.90	$ 68.40	$ 77.80	$ 87.10	$ 96.10	$105.20
0.02	58.50	97.80	96.30	114.40	132.20	149.60	166.70	183.40	199.80
0.03	87.40	114.80	141.40	167.20	192.20	216.50	240.10	262.90	285.10
0.04	115.30	150.70	184.70	217.40	248.80	278.90	307.80	335.50	362.10
0.05	142.70	185.60	226.40	265.10	301.90	336.90	370.10	401.60	431.60
0.06	169.50	219.40	266.30	310.40	351.80	390.70	427.30	461.70	494.00
0.07	195.70	252.10	304.50	353.20	398.50	440.70	474.90	511.70	545.90
0.08	221.40	283.70	341.10	393.90	448.50	492.70	533.30	570.70	605.10
0.09	246.50	314.40	376.20	432.40	483.50	530.00	572.30	630.80	664.10
0.10	271.00	343.90	409.60	468.70	521.90	569.80	612.90	651.70	686.70
0.11	295.10	372.70	441.80	503.80	558.00	606.70	650.00	688.50	722.80
0.12	318.60	400.40	472.40	535.80	591.60	640.70	683.90	721.90	755.30
0.13	341.70	427.30	501.80	566.60	623.00	672.10	714.80	751.90	784.20
0.14	364.00	453.10	529.70	595.60	652.30	707.00	748.10	783.40	813.80
0.15	385.90	478.10	556.40	623.00	679.60	727.70	768.60	803.40	832.90
0.16	407.30	502.20	581.90	648.80	705.10	752.20	791.90	825.20	853.30
0.17	428.30	525.50	606.20	673.20	728.80	775.00	811.30	843.40	870.10
0.18	448.70	548.00	629.40	696.20	750.90	795.80	832.60	862.80	887.50
0.19	468.60	569.60	651.40	717.70	771.40	814.90	850.10	878.60	901.70
0.20	488.00	590.40	672.40	738.00	790.40	832.40	866.00	892.80	914.10
0.21	507.00	610.60	692.40	757.00	808.10	848.40	880.30	905.50	925.40
0.22	525.50	629.90	711.40	774.90	824.50	863.20	893.30	916.80	935.20
0.23	543.50	648.50	729.40	791.70	839.70	876.60	905.00	926.90	943.80
0.24	561.10	666.50	746.60	807.50	853.70	888.90	915.60	935.90	951.30
0.25	578.20	683.70	762.80	822.10	866.60	900.00	925.00	943.80	959.90
0.26	594.80	700.20	778.20	835.90	878.60	910.20	933.60	950.90	963.70
0.27	611.00	716.10	792.80	848.80	889.70	919.50	941.30	957.20	968.80
0.28	626.80	731.30	806.60	860.80	899.80	927.90	948.10	962.70	973.20
0.29	642.10	745.90	819.60	872.00	909.20	935.60	954.30	967.60	977.00
0.30	657.00	759.90	832.00	882.40	917.70	942.40	959.70	971.80	980.30
0.31	671.50	773.40	843.70	892.20	925.70	948.80	964.70	975.70	983.30
0.32	685.60	786.30	854.70	901.20	932.90	954.40	969.00	979.00	985.80
0.33	699.30	798.60	865.10	907.70	939.50	959.50	972.90	981.90	987.90
0.34	712.60	810.40	874.90	917.50	945.60	964.10	976.40	984.50	989.80
0.35	725.40	821.60	884.10	924.70	951.10	968.30	979.40	986.70	991.40
0.36	737.90	832.10	892.70	931.40	956.10	972.00	982.10	988.60	992.80
0.37	750.00	842.50	900.80	937.60	960.70	975.30	984.50	990.30	993.90
0.38	761.70	852.30	908.50	943.30	964.90	978.30	986.60	991.70	994.90
0.39	773.10	861.60	915.60	948.60	968.70	981.00	988.50	993.00	995.80
0.40	784.00	870.40	922.30	953.40	972.10	983.30	990.00	994.00	996.40
0.41	794.70	878.90	928.60	957.90	975.20	985.40	991.40	995.00	997.10
0.42	804.90	886.90	934.50	962.10	978.10	987.30	992.70	995.80	997.60
0.43	814.90	894.50	939.90	965.80	980.60	989.00	993.80	996.50	998.10
0.44	824.40	901.70	945.00	969.20	982.80	990.40	994.70	997.10	998.40
0.45	833.70	908.60	949.80	972.50	984.90	991.70	995.50	997.40	998.60
0.46	824.60	915.10	954.20	975.30	986.70	992.90	996.20	998.00	999.00
0.47	851.20	921.20	958.30	979.90	988.30	993.80	996.80	998.40	999.20
0.48	859.40	926.90	962.00	980.30	989.80	994.70	997.30	998.60	999.30
0.49	867.40	932.40	965.60	982.50	991.10	995.50	997.80	998.90	999.50
0.50	875.00	937.50	968.80	984.40	992.20	996.10	998.10	999.10	999.60
0.51	882.40	942.40	971.80	986.20	993.30	996.80	998.50	999.30	999.70
0.52	889.50	947.00	974.60	987.90	994.20	997.30	998.80	999.50	999.80

TABLE 4—Continued

(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)
Loss in F	Loss on $1,000 when C, the competing ability, is damaged from 2 to 10 degrees more than F, the functional ability								
	C^2	C^3	C^4	C^5	C^6	C^7	C^8	C^9	C^{10}
0.53	896.20	951.30	977.20	989.30	995.00	997.70	999.00	999.60	999.90
0.54	902.70	955.30	979.50	990.60	995.70	998.10	999.20	999.70	999.90
0.55	908.90	959.10	981.60	991.80	996.40	998.40	999.30	999.70	999.90
0.56	914.90	962.60	983.60	992.80	996.90	998.70	999.50	999.80	1000.00
0.57	920.50	965.90	985.40	993.80	997.40	998.90	999.60	999.90	
0.58	926.00	969.00	987.00	994.60	997.80	999.10	999.70	999.90	
0.59	931.10	971.80	988.50	995.30	998.10	999.30	999.80	1000.00	
0.60	936.00	974.40	989.80	996.00	998.40	999.40	999.80		
0.61	940.70	976.90	991.00	996.50	998.70	999.50	999.90		
0.62	945.20	979.20	992.10	997.00	998.90	999.60	999.90		
0.63	949.40	981.30	993.10	997.50	999.10	999.70	999.90		
0.64	953.40	983.30	994.00	997.90	999.30	999.80	1000.00		
0.65	957.20	985.10	994.80	998.20	999.40	999.90			
0.66	960.70	986.70	995.50	998.50	999.50	999.90			
0.67	964.10	988.20	996.20	998.80	999.70	1000.00			
0.68	967.30	989.60	996.70	999.00	999.70				
0.69	970.30	990.80	997.20	999.20	999.80				
0.70	973.00	991.90	997.60	999.30	999.80				
0.71	975.70	993.00	998.00	999.50	999.90				
0.72	978.10	993.90	998.30	999.60	1000.00				
0.73	980.40	994.80	998.60	999.70					
0.74	982.50	995.50	998.90	999.80					
0.75	984.40	996.10	999.10	999.80					
0.76	986.20	996.70	999.30	999.90					
0.77	987.90	997.30	999.50	1000.00					
0.78	989.40	997.70	999.50						
0.79	990.80	998.10	999.70						
0.80	992.00	998.40	999.70						

TABLE 5

Standard of Measurement for determining Damages to F (a) of the body.

Loss in F (a) in anchylosis of				Loss in F (a) in amputation of			
Fingers,		Toes,		Fingers,		Toes,	
Little,	0.01	Little,	0.005	Little,	0.01	Little,	0.005
Ring,	0.02	Fourth,	0.005	Ring,	0.02	Fourth,	0.005
Middle,	0.02	Middle,	0.005	Middle,	0.03	Middle,	0.01
Index,	0.02	Second,	0.005	Index,	0.04	Second,	0.01
Thumb,	0.03	Big,	0.01	Thumb,	0.05	Big,	0.02
Wrist,	0.10	Shoulder,	0.15	Ankle,	0.10	Foot,	0.20
Elbow,	0.15	Fore Arm,	0.25	Knee,	0.15	Leg,	0.25
Hand,	0.20	Arm,	0.30	Hip,	0.15	Thigh,	0.30

TABLE 6—STANDARD OF MEASUREMENT FOR LOSS OF SIGHT.

Standard of measurement for determining damages to F, the functional ability of the body, for the loss of sight in one eye. (Unit g, factor w.)

(1) Degrees of Disability		(2) Scientific Standard	(3) Economic Standard	(4) Loss to	(5) Loss to F.
Slight loss of sight in one eye....	1°	from 0.7 to 0.6	from 6/6 to 5/6	1/6	from 0. to 0.03
	2°	from 0.6 to 0.5	from 5/6 to 4/6	2/6	from 0.03 to 0.06
Severe loss of sight in one eye....	1°	from 0.5 to 0.4	from 4/6 to 3/6	3/6	from 0.06 to 0.09
	2°	from 0.4 to 0.3	from 3/6 to 2/6	4/6	from 0.09 to 0.12
Nearly total loss of sight in one eye....	1°	from 0.3 to 0.2	from 2/6 to 1/6	5/6	from 0.12 to 0.15
	2°	from 0.2 to 0.1	from 1/6 to 0	6/6	from 0.15 to 0.18
Total loss of sight in one eye....					0.18

FOR BOTH EYES.

1° slight loss of sight in one eye with 1° slight loss of sight in the other eye....from 0. to 0.06
1° slight loss of sight in one eye with 2° slight loss of sight in the other eye....from 0.03 to 0.09
2° slight loss of sight in one eye with 2° slight loss of sight in the other eye....from 0.06 to 0.12
1° slight loss of sight in one eye with 1° severe loss of sight in the other eye....from 0.06 to 0.12
1° slight loss of sight in one eye with 2° severe loss of sight in the other eye....from 0.09 to 0.15
1° slight loss of sight in one eye with 1° nearly total loss of sight in the other eye....from 0.12 to 0.18
1° slight loss of sight in one eye with 2° nearly total loss of sight in the other eye....from 0.15 to 0.21
1° slight loss of sight in one eye with total loss of sight in the other eye....from 0.18 to 0.21
1° severe loss of sight in one eye with 1° severe loss of sight in the other eye....from 0.12 to 0.18
1° severe loss of sight in one eye with 2° severe loss of sight in the other eye....from 0.15 to 0.21
1° severe loss of sight in one eye with 1° nearly total loss of sight in the other eye....from 0.18 to 0.24
1° severe loss of sight in one eye with 2° nearly total loss of sight in the other eye....from 0.21 to 0.27
1° severe loss of sight in one eye with total loss of sight in the other eye....from 0.24 to 0.27
2° severe loss of sight in one eye with 2° severe loss of sight in the other eye....from 0.18 to 0.24
2° severe loss of sight in one eye with 1° nearly total loss of sight in the other eye....from 0.21 to 0.27
2° severe loss of sight in one eye with 2° nearly total loss of sight in the other eye....from 0.24 to 0.30
2° severe loss of sight in one eye with total loss of sight in the other eye....from 0.27 to 0.30
1° nearly total loss of sight in one eye with 1° nearly total loss of sight in the other eye....from 0.24 to 0.30
1° nearly total loss of sight in one eye with 2° nearly total loss of sight in the other eye....from 0.27 to 0.33
1° nearly total loss of sight in one eye with total loss of sight in the other eye....from 0.30 to 0.33
2° nearly total loss of sight in one eye with 2° nearly total loss of sight in the other eye....from 0.30 to 0.36
2° nearly total loss of sight in one eye with total loss of sight in the other eye....from 0.33 to 0.36
Total loss of sight in both eyes....0.36

Note.—This table 6, Standard of Measurement for Damages to
F, the functional ability of the body, for the loss of sight, is con-
fined exclusively to the scientific and economic standard of meas-
urement of central acuity of sight, which is the first and most
important indispensable element of vision.

The second indispensable element of sight is the field of vision
which is provided for in Table 6 (a), Standard of Measurement
for Damages to F, the functional ability of the body, for the loss
of any part of the binocular field of vision. The field of vision
embraces half a circle or 180°, divided into six zones of 30° each.
Slight loss of the binocular field of vision embraces its concentric
contraction of 30°, or its equivalent in irregular contraction from
180° to 120°. Severe loss of the binocular field of vision em-
braces its concentric contraction of 60°, or its equivalent in irreg-
ular contraction from 180° to 60°. Nearly total to total loss of
the binocular field of vision embraces its concentric contraction
of 85°, from 180° to 10°, when it becomes total loss of functions
for economic purposes. The damage to F, the functional abil-
ity of the body, is the same for the field of vision, as for the acuity
of vision as given in Table 6 for these designations, that is, for
slight loss of the field of vision it would extend to 0.12; for severe
loss of the field of vision it would extend to 0.24; and for nearly
total to total loss of the field of vision it would extend to 0.36.

The third indispensable element of sight is the muscular move-
ments of the eyes which are classified in Table 6 (b), Standard
of Measurement for Damages to F, the functional ability of the
body, for the loss of the functions of any one or all of the extrin-
sic and intrinsic muscles of two normal eyes. In complete irre-
mediable paralysis of the external rectus of one eye, causing
double vision so the eye had to be excluded from taking part in
binocular vision, the damage to F, the functional ability of the
body, would be the same as the loss of the sight of that eye,
namely, 0.18. In complete paralysis of all the extrinsic and in-
trinsic muscles of both eyes, the damage to F, the functional abil-
ity of the body, would be 0.36.

Provisions are made for all the losses in the field of vision, and
in the muscular movements, which are readily comparable with
the losses in the central acuity of vision.

TABLE 7—STANDARD OF MEASUREMENT FOR THE LOSS OF HEARING.

Standard of measurement for determining damages to F, the functional ability of the body, for the loss of hearing in one ear. (Unit g, factor w.)

(1) Degrees of Disability		(2) Scientific Standard	(3) Economic Standard	(4) Loss to	(5) Loss to F.
Slight loss of hearing in one ear..	1°	from 0.7 to 0.6	from 6/6 to 5/6	1/6	from 0. to 0.02
	2°	from 0.6 to 0.5	from 5/6 to 4/6	2/6	from 0.02 to 0.04
Severe loss of hearing in one ear..	1°	from 0.5 to 0.4	from 4/6 to 3/6	3/6	from 0.04 to 0.06
	2°	from 0.4 to 0.3	from 3/6 to 2/6	4/6	from 0.06 to 0.08
Nearly total loss of hearing in one ear	1°	from 0.3 to 0.2	from 2/6 to 1/6	5/6	from 0.08 to 0.10
	2°	from 0.2 to 0.1	from 1/6 to 0	6/6	from 0.10 to 0.12
Total loss of hearing in one ear..					from 0.12

FOR BOTH EARS.

1° slight loss of hearing in one ear with 1° slight loss of hearing in the other ear........from 0. to 0.04
1° slight loss of hearing in one ear with 2° slight loss of hearing in the other ear........from 0.02 to 0.06
2° slight loss of hearing in one ear with 2° slight loss of hearing in the other ear........from 0.04 to 0.08
1° slight loss of hearing in one ear with 1° severe loss of hearing in the other ear........from 0.04 to 0.08
1° slight loss of hearing in one ear with 2° severe loss of hearing in the other ear........from 0.06 to 0.10
1° slight loss of hearing in one ear with 1° nearly total loss of hearing in the other ear....from 0.08 to 0.12
1° slight loss of hearing in one ear with 2° nearly total loss of hearing in the other ear....from 0.10 to 0.14
1° slight loss of hearing in one ear total loss of hearing in the other ear........from 0.12 to 0.14
1° severe loss of hearing in one ear with 1° severe loss of hearing in the other ear........from 0.08 to 0.12
1° severe loss of hearing in one ear with 2° severe loss of hearing in the other ear........from 0.10 to 0.14
2° severe loss of hearing in one ear with 2° severe loss of hearing in the other ear........from 0.12 to 0.16
1° severe loss of hearing in one ear with 1° nearly total loss of hearing in the other ear....from 0.12 to 0.16
1° severe loss of hearing in one ear with 2° nearly total loss of hearing in the other ear....from 0.14 to 0.18
1° severe loss of hearing in one ear with total loss of hearing in the other ear........from 0.16 to 0.18
2° severe loss of hearing in one ear with 1° nearly total loss of hearing in the other ear....from 0.12 to 0.16
2° severe loss of hearing in one ear with 2° nearly total loss of hearing in the other ear....from 0.14 to 0.18
2° severe loss of hearing in one ear with total loss of hearing in the other ear........from 0.16 to 0.20
1° nearly total loss of hearing in one ear with 1° nearly total loss of hearing in the other ear....from 0.16 to 0.20
1° nearly total loss of hearing in one ear with 2° nearly total loss of hearing in the other ear....from 0.18 to 0.22
1° nearly total loss of hearing in one ear with total loss of hearing in the other ear........from 0.20 to 0.22
2° nearly total loss of hearing in one ear with 2° nearly total loss of hearing in the other ear....from 0.20 to 0.24
2° nearly total loss of hearing in one ear with total loss of hearing in the other ear........from 0.22 to 0.24
Total loss of hearing in both ears........0.24

TABLE 8—STANDARD OF MEASUREMENT FOR THE LOSS OF SMELL.

Standard of measurement for determining damages to F, the functional ability of the body, for the loss of smell. (Unit g, factor w.)

(1) Degrees of Disability		(2) Scientific Standard	(3) Economic Standard	(4) Loss to	(5) Loss to F.
Slight loss of smell	1°	from 0.7 to 0.6	from 6/6 to 5/6	1/6	from 0. to 0.02
	2°	from 0.6 to 0.5	from 5/6 to 4/6	2/6	from 0.02 to 0.04
Severe loss of smell	1°	from 0.5 to 0.4	from 4/6 to 3/6	3/6	from 0.04 to 0.06
	2°	from 0.4 to 0.3	from 3/6 to 2/6	4/6	from 0.06 to 0.08
Nearly total loss of smell	1°	from 0.3 to 0.2	from 2/6 to 1/6	5/6	from 0.08 to 0.10
	2°	from 0.2 to 0.1	from 1/6 to 0	6/6	from 0.10 to 0.12
Total loss of smell					0.12

FOR THE LOSS OF TASTE.

(1) Degrees of Disability		(2) Scientific Standard	(3) Economic Standard	(4) Loss to	(5) Loss to F.
Slight loss of taste	1°	from 0.7 to 0.6	from 6/6 to 5/6	1/6	from 0. to 0.01
	2°	from 0.6 to 0.5	from 5/6 to 4/6	2/6	from 0.01 to 0.02
Severe loss of taste	1°	from 0.5 to 0.4	from 4/6 to 3/6	3/6	from 0.02 to 0.03
	2°	from 0.4 to 0.3	from 3/6 to 2/6	4/6	from 0.03 to 0.04
Nearly total loss of taste	1°	from 0.3 to 0.2	from 2/6 to 1/6	5/6	from 0.04 to 0.05
	2°	from 0.2 to 0.1	from 1/6 to 0	6/6	from 0.05 to 0.06
Total loss of taste					0.06

FOR THE LOSS OF FEELING.

(1) Degrees of Disability		(2) Scientific Standard	(3) Economic Standard	(4) Loss to	(5) Loss to F.
Slight loss of feeling	1°	from 0.7 to 0.6	from 6/6 to 5/6	1/6	Depends upon the extent of the area involved and the functions of the part or parts which have lost the sense of feeling.
	2°	from 0.6 to 0.5	from 5/6 to 4/6	2/6	
Severe loss of feeling	1°	from 0.5 to 0.4	from 4/6 to 3/6	3/6	
	2°	from 0.4 to 0.3	from 3/6 to 2/6	4/6	
Nearly total loss of feeling	1°	from 0.3 to 0.2	from 2/6 to 1/6	5/6	
	2°	from 0.2 to 0.1	from 1/6 to 0	6/6	
Total loss of feeling					do.

No. 9—EVOM TABLE

Based on the premises that F, the functional ability, multiplied by T, the technical ability, multiplied by C, the competing ability, equals to E, the earning ability of a person, and that the gross economic value of man is the present value of all his earnings for a prospective working life, and that the net economic value of a man is the present value of all his earnings less the present value of all his personal expenses for his prospective life.

The money values here given may be used either for the gross, or the net, economic value of man, depending upon whether $1 per day is the gross, or the net, income per day for three hundred days of the year, namely $300 per year, for a prospective working life.

This table is computed on a 3½ per cent. discount basis.

At the age of	Money value	Annual increase	Percentage of increase in 5 yrs.	No. living at	No. deaths in 5 yrs.	Per. of death in 5 yrs.
Birth	133.29			513		
5	1388.44	251.03	911.31	372	141	27.48
10	2900.98	302.50	108.94	355	17	4.57
15	4754.26	370.65	63.88	346	9	2.53
20	5797.72	208.69	24.05	335	11	3.17
25	6114.51	63.35	5.46	321	14	4.17
	Decrease	Decrease	Decrease			
30	5985.71	25.76	2.10	307	14	4.36
35	5664.43	64.25	5.36	291	16	5.21
40	5262.30	80 43	7.13	275	16	5.49
45	4784.29	95.62	9.08	257	18	6.54
50	4178.96	121.04	10.57	237	20	7.78
55	3420.67	151.68	18.14	215	22	9.28
60	2413.00	201.52	29.45	189	26	12.09
65	1141.72	254.25	52.68	156	33	17.46
70	19.10	224.73	98.33	118	38	24.35
75	—609.32	125,68	3293.50	79	39	33.05
80	—1017.88	81.71	67.05	44	35	44.30

(Copyright 1912.)

EXTERNAL RADICAL OPERATION FOR CHRONIC EMPYEMA OF THE AIR SINUSES BY THE ORBITAL ROUTE.*

By W. Sohier Bryant, A. M., M. D.,

New York.

Chronic purulent pansinusitis of the accessory sinuses of the nose is generally admitted to be an affection very resistant to treatment of any kind and practically incurable by any other than surgical means. Because of peculiar development, or because of the extent of the involvement, the surgical access to these diseased cavities lying below the base of the brain at times requires an opening through the upper face, which sometimes results in great disfigurement.

Etiology. Chronic purulent pansinusitis is secondary to neglected acute purulent infections of the nose.

Diagnosis. The diagnosis should be established by the help of a skiagram. There should be an intranasal inspection which, in this conditon, will show pus coming from the orifices of the sinuses and a granular hypertrophic condition of mucous membrane. Transillumination discloses more or less marked opacities in the special areas of the sinuses in proportion to the amount of pus present; and internal and external palpation reveals an edematous condition and hypersensitive areas.

The diagnosis of chronic purulent pansinusitis is easy in cases where the process is advanced enough to be associated with intense continuous or intermittent headache and abundant purulent nasal discharge.

In cases where pain and discharge do not drive the patient to seek relief, the condition may continue unsuspected for years, in spite of grave constitutional symptoms, which indicate toxic conditions with functional nerve disturbances, and cra-

---

*Read at the Annual Meeting of the American Otological Society, at Atlantic City, New Jersey, June, 1912.

nial, heart and lung difficulties. The advance of the process is indicated by severe pain or grave intracranial symptoms.

Surgical Treatment. Our effort in the operation for pansinusitis is to use the technical procedures which give the best results pathologically, functionally and cosmetically. Pathologically, all diseased surfaces must be changed into healthy ones. Pathologic tissues such as pyogenic membranes and granulation tissue obstructing the drainage of the sinuses are to be removed. Functionally, the eye must be restored to its normal position and function, and the nose must again become a moist passage with olfactory sensibility and must contain erectile turbinal tissue. Cosmetically, there must be no external scar, and the lines of the face must not be altered by frontal unevenness and depression.

Surgical treatment is directed to drainage of the sinus affected and removal of pathologic tissue. The external route is chosen in preference to the intranasal route in the cases where the bone conformation does not favor intranasal drainage, in cases where the disease is of very long standing and is far advanced, and especially in cases with dangerous intracranial complications.

These ends are well accomplished by the following technic. General anesthesia is administered by insufflated vapor through a mouth tube.

ANATOMIC OUTLINE.

The usual curved line incision is made through the skin in the center of the eyebrow and down the side of the nose. The cut is carried outward as far as the outer lateral wall of the frontal sinus. Nearly parallel to this cut, but lower down, the periosteum is incised on a curved line which passes along the upper orbital surface and goes farther back, as it passes down the side of the nose. The periosteum is carefully loosened, and lifted with it is the pulley attachment of the superior oblique muscle, and the free part of the lacrimal sac. The eye is retracted by gently drawing the angle of the skin flap downward and outward with a vulsellum forceps.

The bone is opened through the frontal bone at the inner edge of the orbit. The line of bone incision should, if possible, be prevented from crossing the line of the periosteal incision. The object of making these skin, periosteum and bone incisions

on separate levels, is to prevent the stretching of the scar. This technic assures no scar if the sutures hold. Enough of the orbital plate of the frontal bone should be removed so that the frontal sinus may be opened to allow thorough curettage.

We should also excise a part of the nasal process of the superior maxilla, a part of the adjacent nasal bone, as much of the ethmoidal orbital wall as seems necessary to remove all diseased tissues, and the upper part of the plate of the lacrimal bone without injuring the lacrimal sac. The infundibulum should be enlarged as widely as the conformation of the parts will allow, and the middle turbinate and ethmoidal cells should be removed by the aid of clear illumination. Before attacking the ethmoid directly, the procedures are directed to a completion of the frontal sinus work in order to minimize the possibility of postoperative meningitis through injury of the inner table. This is done to prevent infectious material from being carried from a dirty frontal. The excavation proceeds till all cells have been opened and trabeculæ removed. To insure rapid convalescence and to avoid recurrences, this complete removal of cells is as necessary in sinus work as in work on the mastoid cells.

The sphenoid sinus is next opened, and last the maxillary antrum is widely opened above the lower turbinate. These openings are thoroughly curetted and the edges trimmed with alligator punch forceps.

The special instruments used are a medium sized curette with a long malleable shaft for making the initial opening in the orbital plate of the frontal, and a small rongeur to enlarge this opening; the same curette and rongeur are used for cleaning out the frontal sinus; alligator punch forceps are used for removing the middle turbinate. The trabeculæ of the ethmoid are broken down by the curette and trimmed by the alligator forceps. All ethmoidal cells must be broken down in order to assure intranasal convalescence. The curette enters and breaks the wall of the sphenoidal sinus and the alligator forceps trims them smoothly. The maxillary sinus is opened with the same curette; this sinus is cleaned out and the edges are trimmed with alligator punch forceps.

During the operation bleeding is controlled with gauze packing and adrenalin, applied for a few moments as needed. When it is impossible to remove all diseased tissue, a drain is left in

at the inner orbital angle. The wound is sutured by interrupted No. 00 ten-day chromicized catgut for the periosteum, and No. 00 black silk for the skin; a bandage is put across the forehead and a compress over the eye. No dressing is placed in the nose or sinuses or on the external wound, which is bloodless.

Postoperative bleeding is rarely of any moment. If, however, bleeding necessitates postoperative packing, this packing is done with gauze strips placed against the bleeding surfaces through the nose. There should never be indiscriminate packing of the whole cavity.

The advantage of this technic is that no scar remains. There is no possibility of deformed outlines, because of the primary union of the skin wound and periosteum, and because of the preservation of the vertical plate of the frontal bone. Since the operation obliterates all the ethmoidal cells and drains the major sinuses, the convalescence is rapid and there are no recurrences.

Postoperative treatment is directed to keeping up drainage by preventing the occlusion of the orifices of the sinuses, which are necessarily enlarged to their maximum by the operation. Crusts should be softened with hydrogen peroxid, and if granulation tissue is troublesome, it should be removed with forceps. Irritation of the conjunctival sac should be controlled by the use of appropriate collyria. Occasionally a mild boric collyrium or argyrol solution is indicated for use in the eye for a few days. A hydrogen peroxid or oil spray is often needed in the nose to remove exudative accumulations until the mucous membrane has completely covered the denuded surfaces.

Success in this operation depends in large part upon the eradication of all ethmoidal cells, upon obtaining sufficiently large infundibular passage, upon the maintenance of free communication and drainage from all the sinuses, and upon primary union of the external wound.

The surgical technic prescribed for operation on all the sinuses is readily modified to apply to the following groups of sinuses: (1) Frontal, ethmoid, and antrum. (2) Ethmoid, sphenoid, and antrum. (3) Ethmoid and antrum.

The following example illustrates the indications, procedures and results of the operation.

Patient, woman, aged 40 years, with chronic purulent pan-sinusitis. At six years of age patient had scarlet fever and the trouble in the head began then. Since then pus had run from left side of nose. When changing the position of the head, this discharge was at times extremely abundant. Had pain in the left side of nose, over left eye, sometimes in vortex and sometimes in back of head. Intense pain in the head began when the patient had diphtheria at 16 years of age.

There is twenty-four years of definite history of frontal sinus disease following the attack of diphtheria. Fourteen years ago, at 26 years of age, the patient had grippe and intense pain in left eye. At 28 years of age, twelve years ago, she had a severe attack of erysipelas of head and shoulders. Temperature of 105° for nine days. She had phlegmon beginning on top of nose, preceding and after the attack of erysipelas. Has been treated in various cities, then and since. Twelve years ago the patient was not expected to live, was in a comatose condition (probably meningitis) ; not typhoid. No definite diagnosis was made at the time. Patient states that this condition lasted some time. At time of operation the patient had intense pain in left eye, and complained of loud pulsating tinnitus in left ear and of purulent dacryocystitis.

EXAMINATION.

Inspection: Right side of nose negative. Left nasal fossa open, mucosa granular and bathed in pus, chiefly from the middle meatus. Much discharge in nasopharynx. Pulse, respiration and temperature negative. Palpation: Marked tenderness about inner canthus of the left eye, on orbital plate of frontal bone, and above middle meatus in the left nasal fossa. Transillumination: Right side negative. Left side, eye region dark and antral region dark. Showed a large frontal sinus with blurred outline and no boundaries were visible.

Skiagram: Left antrum dense, as if filled with pus. Left ethmoidal and sphenoidal sinuses showed slight increase of density. Left frontal sinus was more obscure than right.

Diagnosis: The above findings indicated sinusitis of frontal, ethmoid, sphenoid and maxillary sinuses.

Operation: Skin was sterilized with Harrington solution. Anesthesia, gas-ether one and one-half hours. Technic fol-

lowed as described above. The mucosa of the frontal sinus was very thick; sinus empty; mucosa of antrum and ethmoids thick; posterior ethmoidal cells contained pus; antrum full of pus. The mucosa of the sphenoid thickened and a little pus was found. Periosteum was sutured with ten-day chromicized catgut No. 00. Interrupted skin suture No. 00 black silk for skin.

<div align="center">CONVALESCENCE.</div>

Day following operation there was some oozing from nose and down throat. Ciliary region of left eye ecchymotic. Relieved some headache by morphin, one-fourth grain, given hypodermically. The temperature reached 100.2° Fahrenheit, rectal. It did not again rise above 99°.

Second day, both eyes closed by edema and ecchymoses about both eyes. Boric acid collyrium was used for left eye, which showed some conjunctival irritation and was slightly purulent.

Third day, removed superficial suture about eye. Took dressing from left eye. Patient had sharp pain on using eye, and diplopia was marked. Much pain on attempting to open left eye. Some pain over top of head. Patient up and dressed. Left nares rather dirty and considerable swelling. Until now the discharge has been clean. Hydrogen peroxid with applicator and oil spray.

Fourth day, all skin sutures removed. Hydrogen peroxid and oil spray in nose.

Fifth day, patient feels very well. Edema nearly gone on right side.

Seventh day, some occipital pain, but no other pain. Patient can open left eye. Nose clear and pretty clean; hydrogen peroxid and oil spray.

Tenth day, no pain, nose felt perfectly comfortable; patient felt well. A little pus from anterior superior region, probably from antrum. Used 10 per cent silver nitrate solution on cotton applicator about nose.

Twelfth day, blowing nose hard had caused a little emphysema about the wound.

Fifteenth day, nose crusty; wiped out.

Sixteenth day, nose clean.

Thirty-eighth day, left eye restored to alignment.

Second month, patient florid, has gained weight. (Since

operation, has had at times various pains, but apparently of
no consequence.) Hearing of left ear has improved very
much. Still slight pulsating tinnitus.

Fourth month, still a slight amount of purulent nose dis-
charge.

Seventh month, owing to the persistence of this slight puru
lent discharge from left antrum, it was necessary to remove a
little granulaton tissue with alligator punch forceps, under
cocain anesthesia. Temperature remained normal after this
operation. Four days later, everything was in good condition
in eyes and nose. No scar remains. The patient has no more
tinnitus. A normal mucosa seems to have been finally estab-
iished in the nose. No syringing or washing was used at
any time by the author, but powders, wiping or sprays were
used. Patient has been under observation one and one-half
years and remains in good condition.

SUMMARY.

The important points of this operation are: (1) The use
of the overlapping flaps. (2) Complete removal of orbital
plate of frontal bone and orbital plate of ethmoidal bone.
(3) Enlargement of infundibulum as widely as the lacri-
mal duct will allow. (4) Complete removal of all cells.

FIGURE I.

Skull marked to show line of incision. x—Skin incision.
xx—Periosteal incision. o—Bone incision.
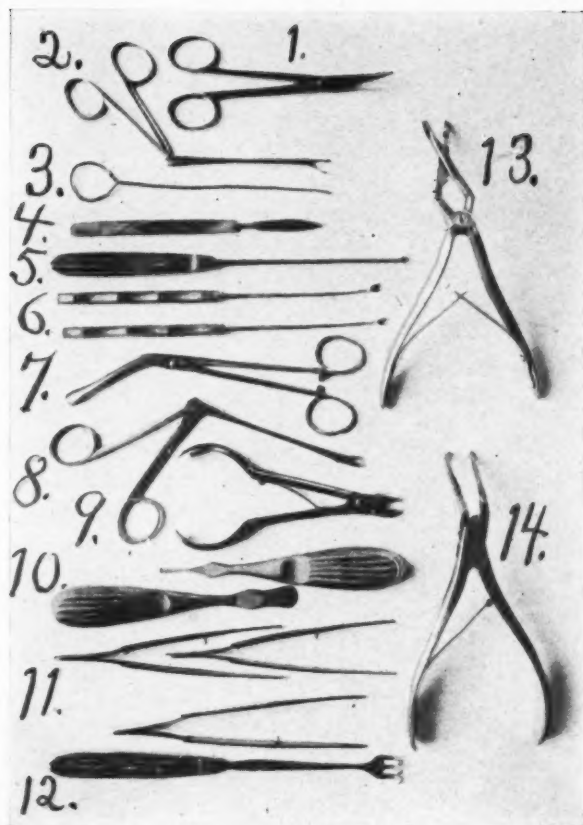
FIGURE II.

Instruments used in the operation besides needle and needle holder. (1) Scissors. (2) Alligator forceps. (3) Probe. (4) Scalpel. (5) Forward cutting curettes. (6) Sidewise cutting curettes with malleable handle, different sizes. (7) Vulsellum forceps, to retract lower flap. (8) Alligator cutting forceps. (9) Short rongeur. (10) Periosteum elevators, different sizes. (11) Forceps, dressing and mouth toothed. (12) Retractor for upper flap. (13) Punch forceps. (14) Longbeaked rongeur.
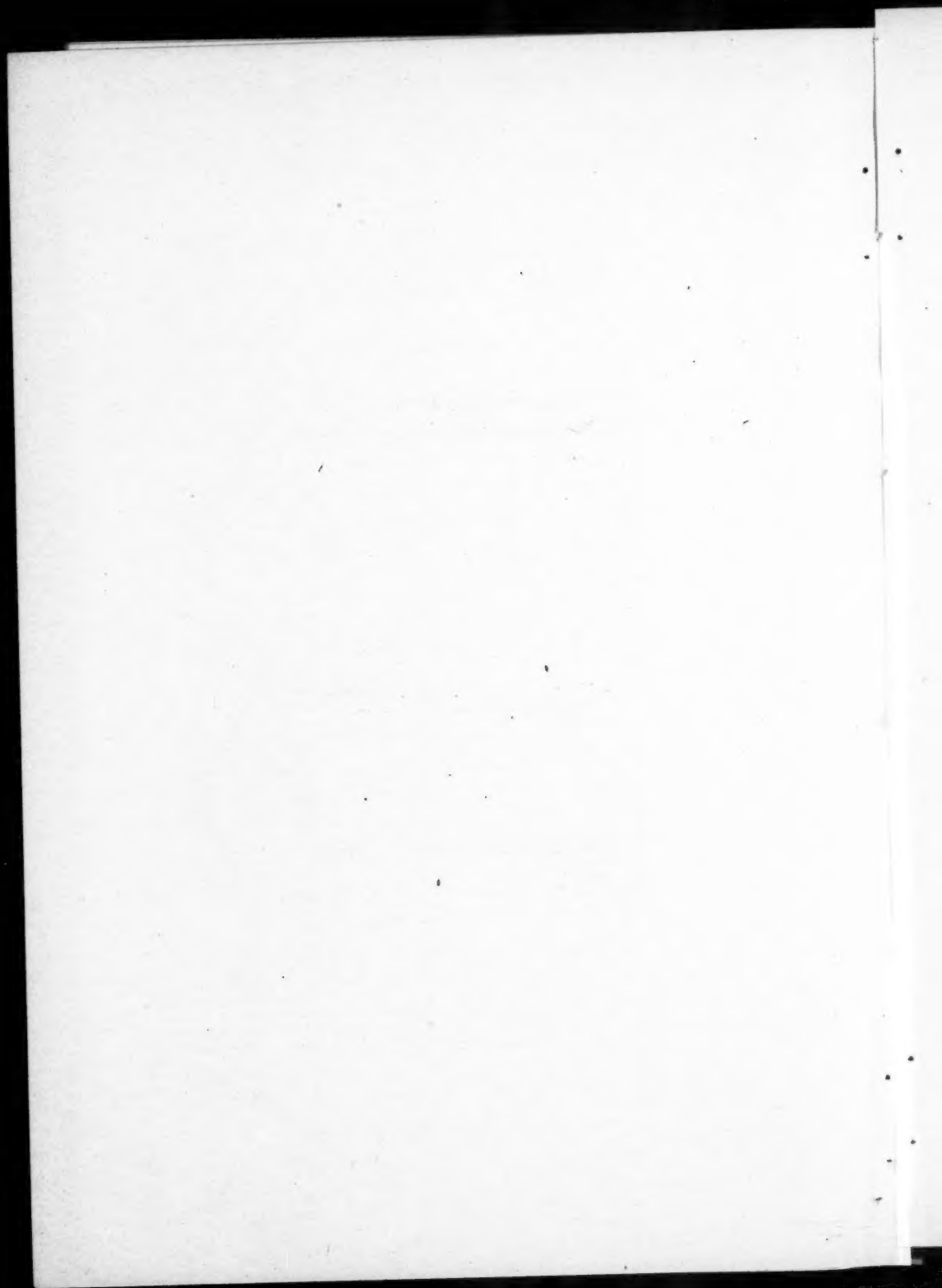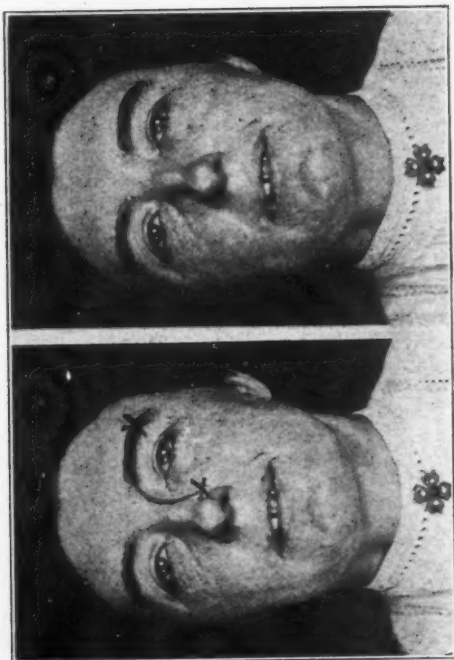
FIGURE III.
Line of incision.

FIGURE IV.
Patient after operation.

LV.

THE NASOPHARYNX IN RELATION TO DISEASES OF THE EAR.*

By Chas. A. Adair-Dighton, M. B., F. R. C. S. Ed.,

Liverpool.

In bringing this subject before you I feel the kind of sensation which, I should imagine, would be felt by anyone rash enough to take coals to Newcastle or fruit to California. I hope, however, that you will be lenient and will treat it in the way it is meant, as a compliment to Dr. Holmes of this city, the introducer of the nasopharyngoscope, and also as an indication that even we in England are beginning to realize that in the nasopharyngoscope we have at once an instrument of as much importance to us in aural surgery as the ophthalmoscope is to our colleagues in ophthalmic surgery.

For generations and generations the nasopharynx has been known as the cause of at least 90 per cent of aural diseases, but owing to its inaccessibility, it has been given scanty attention, and we as aural surgeons have got into a fixed habit of treating the symptoms, the aural complications, and leaving the true diseased area, the nasopharynx, more or less to nature. Up to the last twelve months we have had an excuse, the impossibility of examining the nasopharynx satisfactorily, and the still greater impossibility of carrying out instrumental procedures under the guidance of the eye. We have now no excuse, and it is the purpose of this paper to deal with the nasopharynx, the rock-bottom cause of the aural symptoms so commonly met with in our practice.

Gentlemen, it seems to me best to commence with the diseases of the eustachian tube, the communication between the nasopharynx and tympanum, and I shall divide the diseases of this organ primarily into: (1) Catarrh of the eustachian

*Read before the Ninth International Otological Congress, at Boston, August 12-17, 1912.

tube, or catarrhal salpingitis. (2) Suppuration of the eu-
stachian tube, or suppurative salpingitis. These two main
divisions I shall further subdivide in the course of my paper.

Now, to take up catarrh of the eustachian tube, or catarrhal
salpingitis. This may be at once divided into acute catarrhal
salpingitis, and chronic catarrhal salpingitis. Acute catarrh
of the eustachian tube, or acute catarrhal salpingitis, has been
known from time immemorial as acute middle ear catarrh.
(This is, to my mind, an incorrect name, as it is, after all, and
admittedly, merely the name of the chief symptom met with
in the course of acute inflammation of the eustachian tube.)

The function of the eustachian tubes is to drain and ven-
tilate the tympanum or middle ear. In inflammatory pro-
cesses they become occluded, and the middle ear becomes a
closed cavity. The air, which is blocked up in it and un-
changed, becomes rarefied by the absorption of the oxygen
from it, and a vacuum is created. Due to this vacuum there
is superadded a dilatation of the blood vessels, causing en-
gorgement and hyperemia of the mucous membrane, with
exudation of serum and leucocytes. This causes the usual
symptoms of what has been known as middle ear catarrh, but
what is in reality eustachian catarrh, viz., deafness, pain, tin-
nitus, and a feeling of fullness in the head. These subjective
symptoms are accompanied by objective symptoms. The
pharyngeal orifice of the eustachian tube is red and angry
looking, the posterior wall is swollen, and the floor, posterior
lip, and anterior lip become so edematous as to completely
close it. The ear also shows objective symptoms. In recent
cases the exudation may be visible, the membrana tympani
injected, and the handle of the malleus hidden from view.

This is, I think, the typical picture of acute eustachian sal-
pingitis, and in it we have a picture of inflammation present-
ing exactly the same features as it does when occurring in
other mucous membranes. If in this inflammatory process
the microorganisms gain the upper hand, or secondary infec-
tion is superadded, suppurative salpingitis, or suppurative
otitis media, ensues. With this entity I shall deal later. Pre-
suming, however, that the inflammatory processes triumph and
the swelling, serous exudation, etc., commence, as is usual in
inflammatory processes. to be absorbed, the acute catarrhal
salpingitis may terminate in one of three ways.

Firstly, the condition in which the amount of absorption is perfect, that is to say, one in which the eustachian tube becomes patent, the tympanic structures return to normal, and the auditory symptoms are relieved. This condition, that is, the return to normal, occurs in cases of acute catarrhal salpingitis due to such causes as inclement weather, acute infection of the palatine tonsils, or the exanthemata. Again this result may occur when the cause, such as deflected nasal septum, hypertrophy of the pharyngeal tonsil, enlarged turbinal bones, etc., has been removed.

Secondly, the conditions in which the inflammatory products are insufficiently or inadequately absorbed. In this condition fibrous tissue is laid down in the interstices of the inflammatory tissue, and so bands and adhesions are formed, and fibrous thickening of the parts implicated occurs. The subjective symptoms of this form of chronic catarrhal salpingitis are deafness and tinnitus aurium. These symptoms are associated with a negative Rinne test, a Weber test lateralized to the affected side, and a plus Schwabach test.

THE OBJECTIVE SYMPTOMS.

The pharyngeal orifice of the eustachian tube is swollen and the posterior lip in a tumid condition, serum or seromucous fluid escapes from it, and though it does not appear so angrily red as in the acute condition, it is obviously inflamed. The eustachian tube is occluded and impermeable to normal ventilation. In some cases the fossa of Rosenmuller will be almost entirely obliterated by the approximation of its swollen edges, whilst in other cases bands will be seen stretching across it, and in others the enlarged ends of the inferior turbinal bones will be seen pressing upon the anterior lip of the eustachian tube, whilst again in others adenoid vegetations will be seen overhanging the pharyngeal orifice. The membrana tympani is retracted and the handle of the malleus foreshortened. By inflation and rarefaction by means of Siegle's speculum the drum membrane will be seen either to move in parts, whilst other parts will remain stationary, due to the presence of adhesions binding them down to the promontory, or else to freely flap as a whole. The picture is typical, but you must not underestimate the value of the nasopharyngoscopic examination. The picture of the pharyngeal orifice

and the occlusion of the tube is the clue to a correct diagnosis.

Now, you will ask what is the cause of this condition? It is anything present in the nose or nasopharynx which primarily inhibits respiration and compels mouth breathing. Add to these predisposing factors an acute salpingitis, and the cause is plain. The nasal obstruction causes a vacuum in the nasopharynx which causes a hyperemia and engorgement of the blood vessels, practically the same picture as I have described under the heading of acute catarrhal salpingitis. With these predisposing factors present, catarrh, whether acute or semiacute, will tend to remain and become chronic.

I have described to you the chronic hyperplastic variety, and I believe that in the majority of cases it is the early stage of the third end result of acute catarrhal salpingitis—chronic atrophic salpingitis. It is a matter of common knowledge to all of us that structures that have been in a state of chronic hypertrophy for some time tend gradually to undergo atrophy, and this I think is as true in the eustachian tube as in other parts of the body.

CHRONIC ATROPHIC SALPINGITIS.

In this condition the symptoms are as in the hyperplastic variety, deafness, tinnitus, and in addition the patients have the power of hearing better in a noise—paracusis Willisii. These symptoms are associated with a negative Rinne test, Weber test lateralized to the diseased side, and a plus Schwabach test.

The objective symptoms consist in a widely patent eustachian tube, an atrophied and open pharyngeal orifice, and a pale, tense looking condition of the nasopharyngeal mucosa. The membrana tympani is thin, and on rarefaction with the Siegle speculum may be seen to flap as a whole, almost like a piece of tissue paper. In the fossa of Rosenmuller bands can often be seen, but these are not like the inflamed red bands of the hyperplastic variety, thin tense strands stretching across the fossa.

I have described to you the clinical signs of these two varieties, hyperplastic and atrophic, and have, I hope, shown you that their nasopharyngeal pictures are different. The differential diagnosis between these two conditions is of the utmost importance in the treatment, and there are cases on

the borderline between the two conditions, in which it is hard
to say whether they are of the hyperplastic or atrophic variety.
I am confident that the only diagnostic signs of importance
are as to whether or not the eustachian tubes are patent and
their pharyngeal orifices hyperplastic or atrophic.

The condition of the membrana tympani is often mislead-
ing, as I am certain that this structure shows signs of a change
from hypertrophy to atrophy at a far earlier stage than the
eustachian tubes and their pharyngeal orifices. By this I
mean that it is possible to have a too freely flapping mem-
brana tympani and at the same time a hyperplasia of the
eustachian tubes. By our old methods of diagnosis this was
often overlooked, and the flapping membrana tympani and the
symptoms of paracusis Willisii were looked upon as sufficient
signs upon which to diagnose an atrophic condition. These
conditions certainly are diagnostic of an atrophied condition
of the membrana tympani and the tympanic structures, but
they give no clue to the condition of the rock bottom cause,
the eustachian tube.

These facts have been brought to my notice by practical
experience in the treatment of deafness by Heath's method.
Before the advent of the nasopharyngoscope I was at a loss
to know why some of my cases were completely cured whilst
others were not in the least benefited by this treatment. Since
the advent of the nasopharyngoscope it has been obvious that
those cases that did not benefit were cases in which an atro-
phied membrana tympani was associated with an hyperplastic
eustachian tube. The causation of atrophic salpingitis is the
same as that of the hyperplastic variety, but in addition I think
the old method of inflation, in whatever way used, is responsi-
ble for the change in a great many cases. The cases of an
atrophic drum membrane and an hyperplastic tube are most
certainly due to the evils of inflation. The drum membrane
cannot, naturally, be expected to stand the same force being
applied to it as is necessary to open the eustachian tube, and
so is the first structure to suffer.

Now, to take up the treatment of these three conditions.
First, in acute catarrhal salpingitis the risk of chronic atrophic
salpingitis is far too imminent to allow of the continuation of
treatment by means of inflation, besides which in a state of
congestion the nasopharynx is no longer the sterile cavity it

normally is, and there is the superadded risk of bacteria being inflated at the same time as the air into the tympanum.

My friend Mr. Charles Heath has dealt, or will deal at some length, with the subject of inflation of the ear and its abuses, so that it is unnecessary for me to deal further with it beyond saying that aural surgery has advanced too far to allow this barbarous method to continue to be recognized.

Gentlemen, the common sense treatment of an inflamed surface in other parts of the body is by means of antiseptic and astringent applications to it, and I think that you will find that applications of nitrate of silver or any of its proprietary preparations, such as argyrol, protargol, or sophol, applied directly to the pharyngeal orifice of the tube, are sufficient to cure any case of acute salpingitis not dependent upon demonstrable pathologic lesions for its cause. In cases where there is a deflected septum, enlarged turbinal bones or adenoids, these lesions must of course be immediately corrected.

CHRONIC CATARRHAL SALPINGITIS.

Turning next to the chronic varieties of catarrhal salpingitis, the treatment is somewhat more complicated. In the hyperplastic variety, in which the tube and its orifice are hyperplastic and the membrana tympani is retracted and only moves in parts on rarefaction and inflation by means of Siegle's speculum, the primary treatment must be directed to the tube itself. First of all, all causal factors, such as deflected septa, hypertrophied turbinals and adenoids must be removed. Then the inflammatory condition of the tube must be overcome by means of painting with such drugs as silver nitrate or chlorid of zinc. When the hyperplastic condition has been reduced, attention must be paid to the tube itself, and the patency of this must be restored by the passage of bougies or intratubal injection of the same solutions as are used to paint the pharyngeal orifice, silver nitrate, etc., by means of the Weber-Liel catheter. The ear condition may be entirely left alone, as in cases not too far advanced the auditory symptoms tend to abate spontaneously. In those cases in which the eustachian tube is hyperplastic and the membrana tympani atrophic, the tubes must be the first thought. These must be rendered patent, and when this has been done the symptom, the atrophic drum, can easily be put right by means

of Heath's treatment. Of course, in the hyperplastic as in
the atrophic variety, all bands present in the fossa of Rosen-
muller or polypi, etc., must be removed or broken down be-
fore commencing treatment.

Lastly, turning to the condition in which both the eustachian
tubes and the membrana tympani are in a condition of atrophy.
Again the first thought must be the eustachian tube, but the
treatment of this may be done at the same time as the treat-
ment of the membrana tympani. With a patent and atrophic
eustachian tube and a too freely flapping drum membrane, I
am of a decided opinion that the primary treatment should
be to again tone up the tube by means of stimulating applica-
tions such as iodin, menthol, or hot air, and at the same time
to treat the flapping drum by Heath's method. (I have not
yet had the pluck to paint the tubes with Heath's solution,
but on theoretic grounds this is feasible.) The whole success
or otherwise of any treatment depends on the recognition of
the pathologic conditions to be found in the eustachian tube,
and I sincerely hope I have made these points clear.

My allotted time is drawing near its end, but before closing
I should like to say a word about the suppurative variety of
salpingitis I have referred to.

SUPPURATIVE SALPINGITIS.

The eustachian tube is admittedly the most common, if not
the only, path by which bacteria reach the tympanic cavity, and
yet in all treatments of acute or chronic suppurative otitis
media very little mention is made of it. In cases of acute sup-
purative otitis media, that is, in cases that have not lasted
longer than three to four weeks and have not become compli-
cated by bone infection, a cure can be obtained by treatment
of the eustachian tube combined with the cleansing of the
tympanic cavity and drainage by means of position. The
treatment of the eustachian tube consists in painting it with
silver nitrate, argyrol, sophol, etc., or if it is very severely
implicated, with pus streaming from its orifice, the passage
of medicated bougies. The treatment of the tympanum con-
sists in cleansing it two or three times a day by means of cot-
ton wool mops on the end of long wood probes. Never
syringe a discharging ear.

Then the position can be used to secure drainage. If the

patient is suffering from an unilateral affection with a freely open perforation, he should be made to lie upon the diseased side, so making the perforation the most dependent part of the cavity and the attic and aditus ad antrum the highest part. If this is not done, and the patient lies upon his healthy side, the attic and antrum of the diseased side form the lowest part of that side, forming, so to speak, the bottom of a cistern of pus, and are certain to be affected. Whereas if he lies upon the diseased side, the attic and antrum form, so to speak, the lid of the cistern of pus, and so are comparatively free from the risk of infection. Again, if the patient lies on his healthy side the pharyngeal orifice of the eustachian tube on the diseased side is the most dependent part of that side, and it can easily be seen how bacteria can travel down it into the nasopharynx and infect the pharyngeal orifice, tube and tympanum of the opposite side. The treatment, in short, consists in overcoming the suppuration of the tube by means of applications to it and at the same time keeping the pus drained away from it through the tympanic perforation by means of the posture of the patient. The tympanum meanwhile being dried frequently by means of cotton wool mops.

Chronic suppurative salpingitis, that is to say, one that has lasted more than three or four weeks, is always complicated by disease of the mastoid antrum. The posture of the patient—that is to say, lying on the operated side—will also be found a great help in the after-treatment of these operations, as by it the eustachian tubes are given a rest, the pus and discharge are drained away via the mastoid and perforation in the tympanum, and the tubes, mastoid and antrum gradually but surely return to normal.

THE DURATION OF THE STIMULATION OF THE HAIRCELLS OF THE CRISTA AMPULLARIS COMPARED WITH THE DURATION OF THE ENDOLYMPH CURRENT, AND THE RESULTING NYSTAGMUS.*

By George E. Shambaugh, M. D.,

Chicago.

In the analysis of the physical reaction in the semicircular canals resulting in a stimulation of the haircells of the crista ampullaris, the following facts may be set down as definitely established: first, that the stimulation of the haircells is the result of an interaction between the projecting hairs and the superimposed cupola; second, that this interaction between haircells and cupola is occasioned normally by the impaction of endolymph currents against the sides of the cupola. In this analysis of the stimulation of the haircells of the crista there still remains unsolved the following problems: Is the stimulation of the haircells of the crista of the same duration as the endolymph current, and are the phenomena resulting from the stimulation of the haircells, for example, the nystagmus, of the same duration as the peripheral stimulation?

In attempting to answer these questions Breuer concludes that the duration of the phenomena resulting from the stimulation of the haircells of the crista is the same as the duration of the stimulation of these cells, but that the duration of this peripheral stimulation is much longer than that of the endolymph current which occasions it. In the turning tests, for example, Breuer believes that but a momentary impulse is given to the endolymph on beginning and on stopping the rotation. The fact that the reactions occasioned by the turning tests, both during and on stopping the rotation, last for a considerable period, Breuer accounts for by assuming that the momentary impulse imparted to the endolymph pro-

*Read before the Ninth International Otological Congress, at Boston, August 12-17, 1912.

duces a distortion in the end organ in the ampulla. This distortion consists of a displacement of the cupola on the crista, which produces a stimulation of the haircells that continues until the distortion has righted itself; that is, until the cupola is drawn back to its normal position on the crista. Breuer believes that this is accomplished by the elasticity of the hairs embedded in the cupola, assisted by the contractile action of clumps of mucus lying between the crista and the cupola. In this way, according to Breuer, the reaction occasioned by the stimulation of the haircells, as for example, the nystagmus[1] is of the same duration as the stimulation of the haircells, and both outlast the duration of the endolymph current which produced the stimulation. Breuer's conclusion may be expressed as follows: Duration of nystagmus = duration of peripheral stimulation > duration of endolymph current.

Bárány, in answering these questions, has reached conclusions quite different from those expressed by Breuer. Bárány believes that the stimulation of the haircells has the same duration as the endolymph current which produces the stimulation, but that the reaction following the stimulation of the haircells, that is, the nystagmus, lasts much longer than does the peripheral stimulation. According to Bárány, the duration of nystagmus is not determined by the duration of the peripheral stimulation, but is dependent on the expenditure of stored energy in nystagmus centers. In the turning tests, for example, Bárány assumes that there is but a momentary impulse given to the endolymph on starting and on stopping the turning. This momentary impulse produces but a momentary stimulation of the haircells. This momentary peripheral stimulation, both on starting and on stopping the rotation, sets off each its own nystagmus center, and these require in turn a certain period to expend their stored energy. In this way, therefore, according to Bárány, the duration of the peripheral stimulation is the same as the duration of the endolymph current, while the phenomena resulting from the peripheral stimulation, the nystagmus, lasts much longer. Bárány's conclusions may be expressed as follows: Duration of nystagmus > duration of peripheral stimulation = duration of endolymph current.

In the solution of this problem of the relation between the duration of an endolymph current, the peripheral stimulation

and the resulting nystagmus is found a potent answer to other important problems, as, for example, the question of the existence of nystagmus centers, and the question of the origin of labyrinth tonus, whether in tonus centers or in the haircells of the crista.

This problem of the time relation between an endolymph current, the peripheral stimulation and the resulting nystagmus, is, I believe, capable of a definite solution. I became interested in this problem in the first place through a study of the anatomy of the endorgan in the ampulla. An examination of preparations of this endorgan convinced me that the relation of the cupola to the crista ampullaris is that of a stationary cap fitting over the crista, and not a movable structure, as supposed by Breuer. With the cupola stationary on the crista it became quite clear that any interaction between cupola and haircells, as the result of an endolymph current, must cease the moment the endolymph current ceases. It would seem probable that with the determination of this interaction the peripheral stimulation would also cease. On the other hand, it does not seem impossible, of course, that the activity of the haircells might continue after the interaction between the cupola and the projecting hairs of the haircells had ended. It is quite clear, however, in the light of experiments in the fistula case and the caloric reactions cited below, that such a continued activity of the haircells does not take place. In other words, the duration of the stimulation of the haircells is coextensive with that of the endolymph current.

The step next was to determine the relation between the duration of the peripheral stimulation and that of the resulting nystagmus. Since the peripheral stimulation produced by an endolymph current is of the same duration as this current, one had but to find some way of measuring the duration of an endolymph current. A comparison with the duration of the resulting nystagmus could then be made. In seeking to measure the duration of an endolymph current my first thought turned to the experiment of Ewald, where, after making an opening in the semicircular canal, he was able, by attaching a piston and bulb, to force the endolymph with pressure and suction in either direction through the ampulla. From this experiment it appears that the phenomena resulting from an endolymph current last only so long as does the endolymph current. This experiment suggested to me that

in a suitable case of fistula in the labyrinth a more or less accurate measure of the duration of an endolymph current might be obtained by the compression and rarefaction of air in the external meatus. A case of fistula to be suitable for this experiment should be one where the opening into the labyrinth is not so large but that the compression of the membranous canal will be gradual. With a large fistula the reaction following a compression of air in the external meatus is often so abrupt and violent as to make a careful examination of the resulting phenomena difficult. In order to control the duration of the endolymph current by the duration of the compression of air in the external meatus in a case of fistula in the labyrinth, it is necessary also that the tympanic orifice of the eustachian tube should be closed by a cicatrix.

A case filling these requirements came under my observation about a year ago, and since then I have had repeated opportunities for trying out the experiment I had in mind. The case was one where the opposite labyrinth had been destroyed a few years before through suppuration. The fistula was evidently located in the horizontal canal, since by compression of air in the external meatus a horizontal nystagmus directed to the same side resulted, and by a rarefaction of air a horizontal nystagmus directed to the opposite side was produced. With an end piece which fitted accurately in the external meatus, and a suitable cutoff, I was able, by using a low pressure from a compressed air apparatus, to control, apparently quite accurately, the duration of compression of air in the external meatus as well as the duration of the endolymph current in the horizontal canal.

The results of this experiment were briefly as follows: When the application of compressed air was continuous it required usually about ten seconds before the horizontal nystagmus toward the same side ceased—that is, until the compression of the membranous canal was completed and the endolymph current ceased. Continuing the compression for a longer period produced no further phenomena. On relieving the pressure in the external meatus, it took usually about ten seconds again before the horizontal nystagmus, directed now towards the opposite side, ceased—that is, until the expansion of the membranous canal was completed and the endolymph current stopped.

The interesting feature of the experiment was this: When

the compressed air was applied in the external meatus for
only a couple of seconds and then held, that moment the
nystagmus stopped. By opening and closing the cutoff this
phenomenon could be repeated a number of times before the
moment was reached when the reapplication of the compressed
air failed to produce a return of the nystagmus—that is, until
the membranous canal had been completely compressed. It
was found, also, that the duration of the nystagmus directed
to the opposite side, which followed the releasing of the com-
pression of air in the external meatus, was much shorter when
the compression had been continued for only a few seconds
than when it was allowed to last the full ten seconds. The
result of this experiment demonstrates that the duration of
the nystagmus is the same as the duration of the endolymph
current—that is, the same as the peripheral stimulation. The
conclusion may be expressed as follows: Duration of nys-
tagmus == duration of peripheral stimulation == duration of
endolymph current.

There is still another demonstration of the truth of this
equation. This is a demonstration that can be made with
the caloric reaction. It has been observed that the nystag-
mus resulting from the application of caloric stimulation,
when the head is in a certain position, is altered when the
position of the head is altered. This phenomenon is depend-
ent on the fact that the stimulation of a semicircular canal
resulting from the application of heat or cold depends on
the location of this canal in space. The nearer the canal
approaches a vertical position, the greater the reaction which
can be produced by caloric stimulation, because now the posi-
tion of the canal is best suited to permit of a flow of endo-
lymph by the application of heat or cold. Again, the nearer
the canal approaches the horizontal position in space, the
less active will be the reaction following caloric stimulation,
because now the position of the canal is less suited to permit
of a flow of endolymph from caloric stimulation. When the
canal is in the horizontal plane no flow of endolymph can,
of course, result from the application of heat or cold. Now,
Brünnings has shown that the nystagmus produced by caloric
stimulation while a canal is in a vertical position ceases the
moment the position of the head is altered so as to stop the
flow of endolymph in this canal, which is done by placing
the canal in the horizontal plane, and that the original nystag-

mus will return if the canal is placed again in a vertical position. This phenomena can be repeated as long as any effect of the caloric stimulation lasts. Here we have again a demonstration of the equation given above: The duration of the nystagmus = the duration of the endolymph current = duration of peripheral stimulation.

It is hardly necessary to point out that neither the Breuer nor the Bárány hypothesis offers an explanation for the phenomena observed in these experiments. According to both of these hypotheses, the nystagmus started by the first impulse given to the endolymph should continue for a certain period even after the endolymph current has ceased to flow. In all of the tests, however, the nystagmus stopped the moment the endolymph current stopped.

The chief objections to the conclusion that the duration of the nystagmus is the same as the duration of the endolymph current are: First, the difficulty in accounting for a flow of endolymph in the rotation test that will continue long enough to produce the turning and the after-turning nystagmus; second, the objections pointed out by Bárány against the peripheral origin of nystagmus. These were, the great physiologic difference in the horizontal nystagmus, the fact that at one time the nystagmus to the right, at another time the nystagmus to the left is the greater when tested on different days; the much shorter duration of the rotary as compared with the horizontal nystagmus; the phenomena of the maximum after-nystagmus following ten rotations; and the occurrence of the after-nystagmus following prolonged rotation.

I have discussed elsewhere[2] the first of these objections, namely, the difficulty in accounting for a flow of endolymph in the rotation test that will continue long enough to account for the turning and the after-turning nystagmus. I shall not go into this question here further than to state that I have not found this objection an insurmountable difficulty. While an exact analysis of the physical reactions in the endolymph and perilymph resulting from rotation is impossible, it is quite clear that the duration of the endolymph current in the membranous canals on starting and stopping the rotation is not dependent solely on the inertia of the endolymph in these canals, but is supplemented by other physical reactions resulting from the rotation, which can readily explain the duration of the endolymph current long enough to account

for the duration of the turning and the after-turning nystag-
mus. The difficulty is certainly not of a character to deter
one from accepting a conclusion so clearly demonstrated as
the conclusion that the endolymph current does continue so
long as the nystagmus lasts.[3]

As regards the objections raised by Bárány against the
peripheral origin of nystagmus, none of these constitute funda-
mental objections. Some of these phenomena, to be sure,
are not as yet readily accounted for by a peripheral origin
of nystagmus. On the other hand, neither are they readily
explained by the theory of the central origin of nystagmus.
It seems probable that as we learn to analyze more accurately
the various reactions in the peripheral mechanism the ex-
planation for most of these phenomena will be found here.
As an example of this, take the phenomena of the maximum
after-nystagmus following ten rotations and the occurrence
of the after-nystagmus following prolonged rotation. These
phenomena are not accounted for by Breuer's theory, and
they have been urged repeatedly by Bárány as objections to
the peripheral origin of nystagmus. Bárány has been able
to find in his theory a possible explanation for but one of these
phenomena. This is an explanation for the shorter duration
of the after-nystagmus following five rotations than that
following ten rotations. The explanation Bárány offers is
as follows: It takes about the time to make ten rotations for
a nystagmus center to become exhausted after being stimu-
lated. The center stimulated on starting rotation produces
nystagmus in the direction of the rotation, while the center
stimulated on stopping rotation produces nystagmus in the
opposite direction, the socalled after-nystagmus. If, there-
fore, the rotation is suddenly stopped at the end of ten rota-
tions, that is, at the moment when the center stimulated on
starting the rotation is exhausted, we will get the maximum
after-nystagmus from the center stimulated on stopping rota-
tion. Bárány assumes that in case the turning is stopped
after five rotations, the duration of the after-nystagmus will
be shortened, because now the impulses from the center stim-
ulated on stopping rotation will be in part counteracted by
opposing impulses from the center stimulated on starting
rotation, which has not yet become exhausted. Bárány's
explanation for this phenomena is not entirely satisfactory.

It is not at all clear that the impulses still coming from the center stimulated on starting rotation would, under the circumstances, shorten the duration of the after-nystagmus produced by impulses coming from the center stimulated on stopping rotation. What we might reasonably expect in this experiment, if we accept Bárány's theory of nystagmus centers, is that the after-nystagmus produced by stopping rotation would be weakened until the center stimulated on starting rotation had been completely exhausted. After that time the after-nystagmus should continue exactly as after ten rotations until this second center had become exhausted. In other words, the duration of the after-nystagmus would not be shortened, for it should take exactly the same time for the center stimulated on stopping rotation to be exhausted after five as after ten rotations. The after-nystagmus following five rotations would be weakened for the first few seconds, but the duration should remain the same as after ten rotations. Bárány has not been able to explain with his theory why the duration of the nystagmus following twenty rotations is less than that following ten rotations, nor for the occurrence of the after-nystagmus following prolonged rotation. With my conclusion that the duration of the nystagmus is coextensive with the duration of the endolymph current, all of these phenomena are readily accounted for, as I have shown elsewhere (l. c.), as the result of fatigue from overstimulation of the haircells. These phenomena, therefore, which have been specially pointed out by Bárány as objections to the theory of the peripheral origin of nystagmus, are really additional proofs of the correctness of this theory and of the conclusion expressed above, that the duration of nystagmus = duration of peripheral stimulation = duration of endolymph current. This conclusion is far reaching in its bearing on the physiology of the semicircular canals. It eliminates the necessity for tonus centers to control the duration of nystagmus, and is a potent argument that the origin of labyrinth tonus is in the haircells of the cristæ.

REFERENCES.

1. Breuer did not study the eye movements.
2. Zeitschrift für Ohrenheilkunde, Band lxv, pp. 23-44, 1912.
3. This time relation between duration of nystagmus and endolymph current does not apply, of course, to abnormal conditions.

LVII.

A CONSIDERATION OF OTITIC MENINGITIS IN CHILDREN.*

By S. MacCuen Smith, M. D.,

Philadelphia.

A more accurate title of my paper might be, "A plea for early operation for the prevention and treatment of meningitis and other complications of aural disease," and, as the heading indicates, we will consider, in this paper, only that form of meningitis secondary to aural disease. Increased experience with this affection forces the belief that primary meningitis, per se, is relatively of infrequent occurrence: if it does occur, however, there must be a greatly lowered resistance, due to some predisposing causes, as well as the presence of the exciting microorganisms, which are commonly the pneumococcus or the meningococcus.

Broadly speaking, medicine has not reached the dignity of an exact science, though recent advances in the art of surgical accuracy and precision have placed this branch of medicine well within the domain of scientific exactitude. With such definite knowledge at our command, it seems unreasonable that some of us still adhere to the same hereditary principle of conservatism that characterized medicine in the early days, when all was confusion and skepticism reigned supreme.

The symptoms of acute meningitis are so numerous and their intensity and activity so variable that we must recognize two distinct types: One in which the premonitory stage is characterized by slight but persistent headache, somnolence, malaise, restlessness, increasing irritability and little or no rise of temperature. The other type is distinguished by very acute and severe initial symptoms, the patient usually succumbing to the same in an incredibly short period, whereas

---

*Read before the Ninth International Otological Congress, at Boston, August 12-17, 1912.

the atypical case, of which I have seen two, presents a condition so doubtful in early accurate diagnostic data that I was forced to accept the theory that the aural disease was secondary to the meningeal disturbance. In these cases a cerebral complication was recognized, but an exact diagnosis was impossible.

I have seen some patients present the symptoms of the first type, with slight variation for better or worse, for some days, the ear not being suspected until a discharge was discovered on the pillow.

Headache, frequently the earliest and most persistent symptom, remains localized for a time, but rapidly spreads to adjacent structures when the primary circumscribed lesion becomes diffused. In several fatal cases, briefly considered below, the only symptoms present—and these persisted about one week—were continued moderate headache, a slight temperature rise below 101°, constipation and general malaise. Suddenly the patients presented extreme irritative phenomena, high temperature, rapid pulse, cerebral vomiting, delirium, deepening coma, meningeal cry, Cheyne-Stokes respiration, and death. The age of these five patients ranged between five and eight years. Although the above clinical picture was not absolutely common to them all, they were sufficiently similar to justify their brief consideration together. All had suffered from an acute suppurative otitis media, with spontaneous rupture of the membrana tympani; each had mastoid tenderness on rather firm pressure; two had a suggestion only of redness and swelling over the process; these same two had also drooping of the superior and posterior osseous canal. There was a copious discharge from all, and in each case the pneumococcus predominated. The otorrhea had continued for fifteen days in two cases, and from twenty-one to thirty-two days in the remaining three patients, before terminated by death, and, as above stated, the onset of the fatal issue was sudden and occurred in all five patients in less than fifty-two hours.

The above is a condensed outline of five fatal cases of otitic meningitis, the initial cause of each being attributed to a mild influenzal coryza, the ear receiving little or no consideration until the terminal stage. Furthermore, lamentable as the fact may be, operation was deferred from time

to time, or actually refused, on the assumption that in case meningitis supervened, it is in any case wholly incurable, thus entirely losing sight of effective prophylaxis.

Briefly, then, the underlying cause of the meningitis (the otitis media) was either overlooked or regarded as of little or no importance in the first instance. As the aural symptoms progressed and the meningitis became more and more manifest, and finally fully established, even then, at that late day, the serious suppurative otitis as a causative factor received but scant attention. In other words, in this entire series of cases, not until the terminal complication (meningitis) supervened, did those responsible for their medical care appreciate that the patients were suffering from anything more than what they were pleased to call "a running ear."

Consider the appalling mortality, if such ignorance or irresponsibility were common in the medical profession today, and yet, gentlemen, this is precisely what only too frequently happens in intracranial lesions complicating aural disease, just as peritonitis arising from disease of the intestinal appendix was formerly a prolific cause of death. The latter has been almost entirely eliminated by diagnostic accuracy and prompt surgical intervention, and I trust the time is at hand when we, by means of a more concerted effort, may accomplish equally good results in the treatment of aural complications.

Otitic meningitis is probably our most fatal disease. When the diffuse suppurative type is encountered, it is practically incurable, at present, by means of any known therapeutic measures. The logical conclusion, therefore, is the prompt adoption of every prophylactic agent known to science. Prior to the recent advances in abdominal surgery, appendicular peritonitis was also a very common and fatal disease, but for the past few years, since the advent of modern abdominal surgery, septic peritonitis has become almost extinct. This evolution in the treatment, resulting almost in the banishment of a formerly fatal malady, was made possibly only by adopting the theory that peritonitis was essentially a surgical disease. The logical initial step, then, in the treatment of peritonitis is the prompt removal of the source of the infection—the diseased appendix—and the former will almost assuredly have been prevented. The anatomic arrangement of the ear and its relation to adjacent structures

are precisely similar; indeed, when we keep in mind the frequency with which dehiscences are found, directly exposing the underlying vital structures to the ravages of various forms of infection, we must view aural complications with even greater apprehension. Admitting, then, that appendicular peritonitis is a preventable disease, effective prophylaxis being only possible through prompt surgical intervention, the time has arrived when the profession must also have similar views and adopt like methods in the treatment of otitic meningitis. Furthermore, when diffuse peritonitis is established, it has long since been recognized that our only hope of successful treatment is through operative interference. Otitic meningitis is also preeminently a grave surgical disease, which almost assuredly ends in death when the usual procrastination prevails.

The one distinctive feature that has long since characterized the medical profession is its adherence to the basal principle of conservatism. Doubtless this restraining influence has, in most instances, served us well; and yet many incidents might be cited to show that although reasonable allegiance to the prevailing order of things is not only proper, but highly desirable, on the other hand, both health and life have been only too frequently jeopardized by the adoption of ultraconservatism. Indeed, it would seem that in some instances true conservatism is best served by practicing ultraradicalism, which was aptly termed by the elder Gross, "bold conservatism."

It must be lack of appreciation of the anatomy of the temporal bone, more especially the tympanic cavity, that admits of such lamentable indifference on the part of many of the profession to aural disease. Indeed, the very structure of the mastoid process of the temporal bone favors the collection and promotes the propagation of pathogenic microorganisms, which, in turn, are offered every facility for invading the interior of the skull or entering the general circulation. Were it not for the protective barrier that nature uniformly establishes to restrict or limit the pathologic process, the mortality from secondary intracranial lesions, more especially from meningitis in children, would be appalling. Notwithstanding nature's effort to circumscribe and inhibit pathogenic invasion, the infection is frequently so virulent and overpowering that all her beneficient endeavors are thwarted and an intracranial lesion results.

Even at this advanced stage, however, it is not necessarily too late to institute effectual operative intervention in many cases, if the same is done promptly and resolutely. To my mind, we are not, under any circumstances, justified in waiting for the development of such complications before advising, even urging, an operation as our only means of prophylaxis. Indeed, our responsibility is in no sense modified, nor are we less culpable, even though such procrastination is followed under the guise of socalled conservatism. Granting, then, that our only successful means, at present, of dealing with meningitis lies in our ability to institute efficient prophylaxis, we must necessarily anticipate such complications and resort to early and prompt operative measures as our sole means of preventive medicine in the serious complications of aural disease; indeed, operative otology in such cases stands alone, by reason of its superior excellence over all other methods. It is surprising, even inconceivable, that some aurists still adhere to the ancient methods of procrastination—methods, I may add, that have long since outlived their usefulness, and in many instances are a positive menace to both health and life.

We are reminded that acute mastoid empyema is not necessarily a condition that requires or demands surgical intervention, simply from the fact that we occasionally see cases that empty themselves, and, temporarily at least, result in a spontaneous cure. Instances of this kind would seem to forcibly illustrate the usual exception that proves the rule, for if ever we hope to prevent or cure otitic meningitis or other aural complications, we must treat the initial lesion sanely, promptly and energetically, by whatever means, surgical or otherwise, are best suited to the individual case.

Therefore, I wish to make a plea for timely surgical intervention, not only as an effectual means of preventing aural intracranial lesions (and this is our most important duty), but also as our solitary hope for relief from an existing meningitis. It is claimed by ultraconservatism, and rightly in most instances, that advanced cases of meningitis succumb to the ravages of the disease, regardless of our every effort for its betterment. He who imbibes false comfort from such doctrine, plainly misconceives a fundamental principle, and one of prime importance, that prevention is ever better than

cure. Why, therefore, in the first instance, should we per-
mit a primarily simple aural disease to advance to the ex-
tremity of meningitis, when prompt surgical intervention
would almost assuredly have prevented such a state of affairs?
Then, again, even in extreme cases, made possible as a rule
only by procrastination, when we remember that meningitis is
essentially a surgical disease, we undoubtedly should operate,
and operate at once, and although this may be followed by
death, nevertheless we have given the patient the only remain-
ing chance, and when viewed in the light of recent advances,
this means will, in all probability, become effective in due
course.

If the infective process does not extend beyond the dura, a
circumscribed serous meningitis results, which shows little dis-
position to spread or become purulent, if promptly relieved
by surgical means, but by neglect or delay we court rapid
erosion and consequent perforation of the dura, and subsequent
encapsulated meningeal or brain abscess, or diffuse purulent
meningitis, the latter resulting uniformly in a fatal issue, not-
withstanding our every effort for relief.

The points I have dwelt upon are forcibly illustrated by the
records of the following cases:

Child, A. T., four years of age, was admitted to the Ger-
mantown Hospital, May 24, 1912, suffering from a bilateral
mastoiditis, resulting from an acute otitis media. Until three
years of age the child enjoyed exceptionally good health.
During the year preceding the present illness, he had whoop-
ing cough and several attacks of tonsillitis.

Three days before the patient was admitted to the hos-
pital, he was seen by the attending physician on account of
severe pain in the right ear, from which he had suffered for
two days previous, and was given internal medication, with
instructions to irrigate this ear. The parents state that the
child had a chill on the second day of its illness, lasting
about an hour, and they also noticed that the left auricle pro-
jected from the head, although there was no visible swelling
back of the ear.

The baby was seen again in three days by the attending
physician, who observed what he termed "a meningeal facial
expression," and directed that the patient should be taken to
the hospital at once. When admitted, both ears were dis-

charging, the drum membranes having spontaneously rupt-
ured, and there was distinct tenderness on pressure over each
mastoid process. The child's mental state was decidedly dull,
and it gave expression to its suffering by the frequent utter-
ance of the meningeal cry.

A lumbar puncture was done, which showed the fluid
cloudy and containing pus. A differential blood count gave
leucocytes 35,000 and polymorphonuclears 75 per cent.

A double simple mastoid operation was at once performed,
it now being only six days after the initial earache and three
days after the first appearance of the discharge. The necrotic
process was very complete, free pus escaping when the
incision in the soft parts was made, through a large carious
opening in the cortex. On each side there was a perisinus
abscess formation extending almost the entire length of the
vessel, although the latter was well covered with a protec-
tive coat of granulations and did not seem to be otherwise in-
volved. There was free communication between the mas-
toid antra and the external auditory canals. At the time of
the operation, the child's temperature was 105.4°, declining
to 104° following the operation. The next morning it dropped
to 100°; at 1 p.m. it was 101°, and at 9 p.m. 104°. The
patient remained very irritable and contiued to give its fre-
quent meningeal cry. The pupils reacted to light and there
was no discoverable change in the fundi. The head was dis-
tinctly drawn backward, and there was apparently some loss of
motion in the upper extremities. Immediate operation was
advised for the relief of intracranial pressure, and this be-
ing agreeable to the parents, we performed the Haynes op-
eration for providing drainage through the cisterna magna.
The intracranial pressure was very marked, as was shown by
the fluid escaping in a stream several inches high, through
the small opening we made in the dura. Unfortunately, the
patient died, but I wish to commend this operation, as de-
vised by Prof. Irving S. Haynes, of New York, for the estab-
lishment of intracranial drainage, as probably the best that
has yet been offered. There is no question whatever as to
efficient drainage being obtained, by this method, though
I believe that up to the present time no case of meningitis
has been seen sufficiently early to warrant any definite con-
clusion as to its actual utility.

An examination of the cerebrospinal fluid withdrawn at the time of operation showed it to be cloudy and to contain colonies of staphylococcus pyogenes albus.

The outcome in this instance shows how rapidly meningitis can develop, and also forcibly illustrates the importance not only of early recognition of acute ear disease, but also the vital necessity for surgical evacuation of tympanic secretion by means of a free incision of the membrana tympani, in contradistinction to permitting spontaneous rupture.

I am indebted to Dr. Frank B. Gummey, of Philadelphia, for the history of the following interesting case:

Baby S., age ten months, had always been a robust and healthy child until it developed croupous pneumonia. Six days after the onset of this infection, the child began to suffer from pain in the right ear, shown by placing the hand over that ear, and two days later there was distinct swelling and fluctuation over the mastoid process, without otorrhea. I saw the child at this time, but as it was at the height of its pneumonia, I decided not to operate until after the crisis, which occurred twenty-four hours later. The patient was then taken to the hospital, where a simple mastoid operation was performed, followed by an uneventful convalescence and complete recovery in fifteen days.

The child remained perfectly well for seven months, when it was taken sick with an attack of grippe. The temperature ranged from 102° to 104°, and the baby cried continuously for two days, when a discharge of pus appeared from the opposite (left) ear.

I was called at this time, and as the symptoms were not urgent, I advised irrigations and careful watching, but the next morning the child's mentality and general condition were so much worse that we decided upon immediate operation and the patient was removed to the hospital, where a mastoid operation was performed. The necrosis was very extensive but no communication with the interior of the skull was discoverable. The patient grew steadily worse, and two days later had two convulsions, the head was drawn back, and it presented additional symptoms of meningitis. At this time, also, examination of the chest revealed rales over the base of both lungs, the temperature mounting to 107°.

Notwithstanding further operative intervention for the re-

lief of intracranial pressure, the child suffered from two more convulsions, dying eight hours later, or on the fourth day of the disease.

The above history shows, in the first instance, what seemed to be a primary pneumococcic mastoiditis, for although the tympanic cavity may have been the original site of the disease, it gave no evidence of the same when first seen by me. Examination of the pus evacuated from the mastoid process showed a pure pneumococcic infection. It is worthy of note that the patient enjoyed excellent health for a period of seven months before the opposite ear became involved in an acute suppurative otitis media, with simultaneous mastoid suppuration and spontaneous rupture of the membrana tympani. Here, again, the pneumococcus was shown in pure culture, both in an examination of the pus and lumbar puncture.

A CASE OF SEPTIC THROMBOSIS OF THE LEFT SIGMOID, LEFT CAVERNOUS, AND LEFT INFERIOR PETROSAL SINUSES, WITH A SUGGESTION FOR TREATMENT IN FUTURE CASES.*

BY

CHARLES A. BALLANCE, M. V. O., M. S., F. R. C. S.,

AND

NEILL HOBHOUSE, B. A., OXON.,

LONDON.

A boy, aged twelve years, was admitted to St. Thomas' Hospital on April 29, 1912, with discharge from the left ear and tenderness, swelling, and pain behind the left mastoid.

Previous History.—Scarlet fever at the age of two years: subsequently measles and chicken pox. Left ear has been deaf, and there has been occasional discharge from it, since scarlet fever. A throat operation was performed when patient was three years of age. Adenoids removed four weeks ago.

History of Present Illness.—For some months has had occasional pain in and around left ear. One week ago severe pain in ear occurred, and more discharge was noticed from the ear. The pain has continued, and patient has been drowsy for three days. No vomiting. Complains of pain in back of head and neck on sitting up.

State on Admission.—Left pinna displaced downwards and outwards by a mastosquamous swelling which is very tender. Foul pus in left ear. Hearing nil in this ear except by bone conduction with tuning fork on skull. Lies curled up in bed on right side. He is drowsy and apathetic Pupils normal. Horizontal nystagmus in both directions, most marked in left

---

*Read before the Ninth International Otological Congress, at Boston, August 12-17, 1912.

eye. No retinal changes. Right knee jerk more brisk than left. Plantar reflexes flexor. Slight ataxia in walking. Temperature, 102°; pulse, 108. Spleen easily felt. The left external jugular vein is very prominent, and a large vein extends over the left side of the scalp, from the region of the parietal foramen to the upper part of the external jugular.

Operation forthwith. Bone behind ear exposed by vertical and horizontal incisions. The complete mastoid operation was done. Every cell in the mastoid lined with grayish white gangrenous mucous membrane. Not a great deal of pus was found in the mastoid, but a cholesteatomatous mass occupied the tympanoantral cavities. The sigmoid was then explored. Pus oozed from the bony groove of the sinus. The sinus was exposed for a considerable distance, and the dura, in front of and below it, over a considerable area, was also exposed. The outer layer of dura bounding the sinus for two inches was white and gangrenous. On removing it no bleeding occurred, as the sinus was thrombosed. The dura in front and behind the sinus was white for about one inch, its outer layer being evidently dead. The sinus was followed backwards nearly to the torcular. The petrous in front of the sinus was cut away till the back of the bulb was exposed. In doing this the facial canal was defined. The jugular vein was then exposed in the neck, its tributaries tied, and after being divided between ligatures the upper end was brought to the surface (Fig. 1). Lumbar puncture yielded one ounce of opalescent fluid.

May 1st.—Temperature 104°, tongue dirty, patient drowsy. Lateral sinus, bulb, and upper segment of jugular irrigated with hydrogen peroxid. The stream of fluid through the bulb was quite free. This irrigation was done daily when the dressings were changed. The cultures made at the operation showed a growth of bacillus pyocyaneus and staphylococcus aureus. A vaccine was prepared.

May 7th, 2 p. m.—Patient has remained much in the same state since last note, with a temperature varying from 102° to 105°. This morning at 9 a. m. temperature was 107°, and edema of the left upper eyelid and slight proptosis of the left eye was noticed. Now at 2 p. m. these signs are the same and there is some restriction in the movement of the left eye outwards. Pupils react normally. No change in optic discs, and no engorgement of retinal veins. Spleen larger. Involuntary

tremors in legs. Well marked patellar clonus on each side. No heart murmurs.

May 8th, 10 a. m.—Edema of left eyelid, as before, but there is now chemosis of the conjunctiva external to the cornea. Left pupil almost stabile. Movements innervated by left third nerve, all affected, and no outward movement of left eye is possible. Retinal veins engorged. Mr. Lawford (the ophthalmic surgeon) suggested that the absence of retinal signs yesterday might have been due to the thrombosis only affecting at first the posterior part of the cavernous sinus, so that the retinal blood was able to escape freely by means of the pterygoid plexus. Right eye normal.

Operation forthwith. The left cavernous sinus was exposed by the Hartley-Krause incision for operation on the Gasserian ganglion. No trouble on account of hemorrhage was met with. An incision into the cavernous sinus was made about one inch long, and out came blood clot, and pus from the posterior end of the incision. (Fig. 2.) After the cavernous sinus had been irrigated with hydrogen peroxid a tube was passed into it and fixed there.

Attention was then given to the wound made at the previous operation behind the ear. It was found that the dura in front of the vertical portion of the sinus was gangrenous in its whole thickness, and from the upper and inner part a little pus mixed with blood was oozing from an opening in the sloughed dura. This opening was enlarged and the dead dura removed, when a meningocortical abscess of the cerebellum about the size of a Barcelona nut came into view. This was washed with hydrogen peroxid, and tamponed with a strip of gauze.

May 9th.—Rigor. Temperature, 107° at 8:30 a. m.; pulse 130. Slight edema of right upper eyelid. No retinal changes in right eye. Incontinence of urine. Slight swelling and tenderness on dorsal aspect of left wrist. Vaccine given. The cultures taken from the septic clot removed from the cavernous sinus grew a streptococcus.

May 10th.—Besides slight edema of right upper eyelid there is slight impairment of movement in right external rectus. There are no retinal changes in right eye. Temperature, 105°; pulse, 140. Patient died this afternoon.

AUTOPSY.

Part of the left lateral sinus, towards the torcular, not exposed in wound, contained uninfected clot. Bulb and upper segment of left jugular free of clot. Left inferior petrosal sinus full of pus. Left cavernous sinus empty except at posterior part, where a little pus was seen. Left superior petrosal sinus normal. Left half of circular and left half of transverse sinuses contained septic clot. No sign of disease was found in the right cavernous sinus. The meningocortical abscess cavity was free of pus. It had been satisfactorily drained and tamponed. The brain and other organs showed no further pathologic changes except those due to high temperature.

REMARKS.

Thrombosis of the cavernous sinus may occur from frontal sinus or sphenoidal sinus infection, from cellulitis of the face, carbuncle of the neck, meningitis, sarcoma of the base of the skull, marasmus, and traumatism, as well as from extension of septic processes from the signoid sinus or petrous bone. Dwight and Germain,[1] in an important paper published in 1902, refer to 184 published cases in which only fourteen recovered. In one of Dr. Dwight's cases he incised the cavernous sinus after opening the dura of the middle fossa. In this series of cases, forty-three were ear cases, and in all the sigmoid sinus was involved primarily.

Knapp[2] had previously published a case of aseptic thrombosis of the cavernous sinus in which operation was done by Hartley. The thrombosis was due to the growth of a sarcomatous tumor. Aseptic clot was removed, but Meckel's space was opened. Meckel's space certainly ought not to be incised in septic cases, as such a procedure opens the subarachnoid space. The operation was done on March 1st, and death occurred on May 16th. The operation, so far as it concerned the cavernous sinus, was successful.

Knapp suggests that the operation should be extended to septic cases, but Bircher had operated successfully on a septic case eight years previously.

St. Clair Thomson,[3] in a paper on the cerebral and ophthalmic complications of sphenoidal sinusitis, deals with cases in which the cavernous sinus has become infected from disease

lying either in front or below it. In the cases with which this
paper deals the sinus is always affected first at its posterior
extremity.

Levinger[4] of Munich has recently recommended for infective
thrombosis of the cavernous sinus the removal of the contents of
the orbit and resection of posterior part of the bony inner wall of
the orbit up to the optic foramen. This method allows of the ex-
posure of the inner and lower wall of the cavernous sinus after
a further removal of bone has been done, namely, the resection
of the outer and anterior boundaries of the sphenoidal sinus.
This method has not been tried on the living. It would appear
that the throwing of the optic foramen and sphenoidal fissure
into one channel by removing the bone between the two would
effectively expose the anterior end of the cavernous sinus with-
out touching the sphenoidal sinus.

Treatment that is desirable in septic thrombosis of the
ophthalmic vein and anterior end of cavernous sinus, such as
clearing out the contents of the orbit and incision of the an-
terior end of the cavernous sinus, is clearly inapplicable to an
infection which has spread from the bulb by way of the in-
ferior petrosal sinus to the posterior end of the cavernous
sinus. The cause of the infection of the cavernous sinus being
determined, the appropriate operation will be obvious, as the
infection is one continuous process which should be followed
from its primary site to its ultimate extension.

In 1887 Coupland[5] described a case of thrombosis of the
cavernous sinus and gave the following clear description of
the symptoms which may appear in this disease:

1. Those due to venous obstruction. (a) Proptosis. (b)
Edema of eyelids and chemosis of the conjunctiva when the
thrombosis extends into the ophthalmic vein. (c) Edema of
the face when the facial vein is thrombosed. (d) Enlargement
of the frontal veins, due to diversion of the circulation through
the orbitofacial anastomosis. (e) Venous hyperemia of the
retina and choked disc are not symptoms of thrombosis of the
sinus. Such congestion depends upon the obstruction involv-
ing the ophthalmic and retinal veins.

2. Those due to interference with nerves. (a) Supraorbital
neuralgia, due to irritation of the first division of the fifth
nerve. This division of the fifth nerve is generally the first to
suffer. (b) More or less paralysis of the muscles of the eye,

which may amount to complete ophthalmoplegia, due to affection of the third, fourth and sixth nerves. The completeness of the ophthalmoplegia indicates a change in the wall of the sinus as well as in its contents, i. e., phlebitis as well as thrombosis.

The remaining remarks refer exclusively to infection of the posterior end of the cavernous sinus in temporal bone suppuration.

The cavernous sinus may be primarily and directly infected from the bone disease when this has extended to the apex of the petrous, the infective focus in the sinus is then directly continuous with that in the bone; in other cases infection reaches the cavernous sinus by way of a connecting sinus. In the case of Bircher[6] the operator worked his way through the petrous until foul pus escaped from the posterior end of the cavernous sinus. For approaching the cavernous sinus in these cases I have adopted the Hartley-Krause method for exposing the Gasserian ganglion, and found it easy and effectual, but probably when pus has been evacuated from the sinus, it would be well to adopt the recommendation of Voss[7] who cuts away the zygoma and removes more bone from the basal aspect of the skull so as to get direct drainage. Operations on the cavernous sinus must be done at an early stage of the infection, because of the great facility with which the disease extends to the opposite sinus through the circular and transverse sinuses, and also because the meninges are likely to be infected. A few years ago (October, 1907) I operated on a boy of six years who had septic thrombosis of the cavernous sinuses following suppuration in the right temporal bone. The right cavernous sinus was easily opened, and on incision of it a teaspoonful of pus escaped. A drainage tube was fixed in the sinus, but the patient died twenty-four hours later.

Passow[8] has quite recently published a case of fatal sinus pyemia in which the inferior petrosal contained a septic clot and was the only sinus obviously affected. It is probable that these cases are, like cases of isolated thrombosis of the bulb, more common than has been hitherto recognized.

The case recorded in this paper is of great interest because careful observation was possible of the signs and symptoms of the onset and progress of the cavernous sinus thrombosis. The operation successfully dealt with the septic

thrombosis of the sigmoid and cavernous sinuses: that on the cavernous sinus was done early enough to prevent extension to the other side. There was nothing found at the postmortem examination to explain the occurrence of slight edema of the right upper eyelid. The operation, however, did not arrest the acute septicemia from which the boy was suffering. The inferior petrosal sinus remained untreated. It was the pathway of infection from the bulb to the cavernous sinus. The current of blood in the inferior petrosal sinus is towards the bulb, but septic venous infection can easily extend in a direction contrary to a blood stream so sluggish as that in the inferior petrosal sinus. Recognizing that the local infective process is continuous, and that the whole extent of the local infective process demands surgical intervention, it is clear that the operations performed on this case, though extensive, were yet not adequate to arrest the absorption of infective material. When the cavernous sinus was found infected, not only should it have been freely opened and drained, as was done, but the bulb of the jugular should have been freely exposed and laid open so that the opening of the inferior petrosal sinus into the bulb could be seen, and irrigation of the inferior petrosal sinus carried out from the bulb to the cavernous sinus. This is the plan on which I propose to proceed in any future case of infection of the cavernous sinus from the bulb that may come under my care.

In cases of osteomyelitis of the petrous, the only treatment is to remove it piece by piece, as did Bircher: irrigation of one or both petrosal sinuses would in such circumstances be insufficient to save life. The cutting away of the whole petrous must mean, too, the exposure and, if thought desirable, the deliberate opening of the petrosal sinuses. The additional advantage is gained of bringing the posterior end of the cavernous sinus directly into the field of operation.

It is not necessary to discuss the best method of completely exposing the bulb. Operating surgeons have devised various plans to this end. The papers of Grunert, Piffl and Voss on this subject are well known. In my opinion there is no better plan than that of following the sinus till the bulb is reached. The subsequent removal of the bony external boundary of the bulb should present no real difficulty. It should be remembered that the bulb varies considerably in size and in its exact rela-

tion to the tympanum. It sometimes attains the size of a large cherry and is sometimes scarcely noticeable. I have on several occasions exposed and incised the bulb in septic cases without meeting with any considerable difficulty and without injury to the facial nerve.

ADDENDUM.

The following cases referred to in this paper are here given in more detail:

1. Bircher's Case.—Female, aged twenty-five years. In 1877 had bilateral aural suppuration following scarlet fever. September, 1892, fever with rigors. Intense headache. Tenderness over left mastoid and halfway down the neck along the jugular. Mastoid operation September 8th. Pus in sinus groove. Great improvement for three days. Then remittent fever up to 104°. September 16th, palsy of left third nerve, with irritation of first division of fifth (pain in frontal region). September 19th, sixth and fourth nerves also paralyzed so that the ophthalmoplegia became total. September 20th, two trephine holes made above meatus, pyramid removed piecemeal, until only the lower wall of the carotid canal was left. No further collection of pus was found until the apex was removed, and then a gray discolored spot was seen from which stinking pus was coming. The patient made a good recovery, but with permanent ophthalmoplegia and a facial palsy.

Remarks.—In this case the posterior end of cavernous sinus or the inner extremity of the inferior petrosal sinus was opened, and hence efficient drainage was obtained.

2. Passow's case of infection from inferior petrosal sinus.— Six weeks after the wound from an operation for acute mastoid disease had healed, tonsillitis and pyemia with metastases supervened. The pyemia was at first attributed to the tonsillar infection, but on optic neuritis being found the mastoid wound was explored, the sigmoid sinus was exposed and opened, but nothing abnormal was found. At the autopsy a septic thrombus as large as a pea was found in the inferior petrosal sinus. Passow suggests that the clot was at first noninfective, but was secondarily infected from the tonsil.

Remarks.—This case illustrates the importance of the inferior petrosal sinus as a source of infection.

3. Coupland's case was a woman forty-three years of age. The disease commenced with severe left supraorbital neuralgia

and drooping of the left lid. The illness lasted from November to March. Towards the end of January the right lid drooped and there was severe right supraorbital neuralgia. Finally on both sides proptosis, ophthalmoplegia, chemosis of the conjunctiva, which was insensitive, occurred, but the optic discs and retinal grounds remained normal. There was no edema of the eyelids or any enlargement of the facial veins. The autopsy revealed basal meningitis. The left cavernous sinus was occluded by nonpurulent clot, but the right cavernous, circular, and transverse sinuses contained purulent clot. The petrosal sinuses were empty. The source of the infection was not discovered.

Marasmic thrombosis occurs at the two extremes of life. Coupland gives some examples. He also relates three interesting cases of thrombosis of the cavernous sinus due to trauma: (1) Jordan Lloyd's case following fracture of the base of the skull; (2) Pitha's case of mastoid necrosis after a sabre cut, leading to thrombosis of the lateral and cavernous sinuses, and (3) Halke's case, which followed a blow on the head and simulated orbital aneurism. The paper concludes with a table of twenty-eight cases.

4. C. A. Ballance's Case.—The patient, a boy, was admitted to St. Thomas' Hospital on October 17, 1907, in a drowsy condition with suppuration of the right mastoid region. The temperature was 103°, pulse 104. He had been ill a little more than one week. There was proptosis of the right eyeball, edema of the right upper lid, and paralysis of the movements of the right eyeball. The same condition was commencing on the left side: there was slight proptosis and slight edema. On the right side the retinal veins were large and turgid, but there was no change in the optic disc. The left fundus was normal. The complete mastoid operation was done forthwith on the right side and an incision was made into the right cavernous sinus. Death occurred thirty hours after the operation.

Autopsy.—Basal meningitis. Dura over tegmen on right side adherent and inflamed. Right temporosphenoidal lobe adherent and softened over tegmen. Right petrous discolored, necrotic and infiltrated with pus. Septic thrombosis of both cavernous sinuses, the right sinus being more diseased than the left. Circular sinus full of pus. Right sigmoid sinus and jugular vein unaffected. There is no note in the postmortem record of the state of the right petrosal sinuses.

Remarks.—This was a case of suppuration of the petrous and may be compared with Bircher's case. The infection of the right cavernous sinus may have been due directly to the acute osteomyelitis of the apex of the petrous, without the intervention of either the inferior or superior petrosal sinus.

5. Lombard's Case.[9]—A French foot-soldier who had had several attacks of malarial fever was taken ill on April 1, 1903, and got worse from day to day. On the 6th he began to have fever with remissions, the attacks of fever at first occurring at regular intervals, about 5 a. m. and 6 p. m. Subsequently they became irregular. On April 9th herpes appeared around the mouth and nose; pneumonia was at first thought of, but no physical signs of it appeared. On April 11th he was sent to hospital. On admission he was quite prostrated, had intense headache and a dry tongue. Fever up to 104° with large oscillations. He was treated for five days as a case of malaria. On April 16th swelling of the right eyelid was noticed, and there was an erysipelatous rash over the limbs and part of the trunk. The right eye became prominent and tender, and the pupil ceased to act to light. The edema and proptosis increased. Suppurative otitis was found on the right side. The frontal sinus was opened, but nothing abnormal was found. A few days later the patient died comatose.

Autopsy.—Infective thrombosis of right cavernous sinus and suppurative meningitis. No visible track connecting the aural disease with the cavernous sinus was found.

Remarks.—Edema of the eyelid may occur in frontal sinus suppuration, but since there was already suppurative otitis, it would seem that the temporal bone suppuration should have been dealt with at once.

6. Poulsen's Case.[10]—Male, aged five years. Discharge from right ear one year. Admitted to hospital July 3, 1884. He had been ailing fourteen days with headache, fever and pain in the right ear; he had become apathetic and fretful; the bowels had been confined. He had not vomited. On admission apathetic, but screamed on the least touch. Temperature, 102.2°. Pupils equal but sluggish. Slight discharge from right ear. Next day cyanotic and vomited frequently, often screamed loudly. The day following he died.

Autopsy.—Pus in tympanum. Extradural abscess in sigmoid groove. Thrombosis of superior and inferior petrosal

sinuses. No meningitis. Brain normal. Superior longitudinal sinus and lateral sinus normal. Dura easily detached from petrous nearly to the internal auditory meatus; here a little pus was found and the superior and the inferior petrosal sinuses were blocked by thrombi.

Remarks.—This case, like that of Passow, is an example of isolated thrombosis of the petrosal sinuses. No temporal bone operation was done. The case occurred many years ago. At the present time the temporal bone disease would be promptly dealt with, and the sigmoid groove abscess and that by the internal auditory meatus would be evacuated by following the pathway of infection. Modern surgery offers a fair prospect of saving the lives of such patients.

REFERENCES.

1. Dwight and Germain: Boston Medical and Surgical Journal, 1902.
2. Knapp: Archives of Ophthalmology, 1900.
3. Thomson, St. Clair: British Medical Journal, September, 1906.
4. Levinger: Zeitschrift für Ohrenheilkunde, Vol. 64, 1912.
5. Coupland: Transactions of the Ophthalmological Society, Vol. VII, 1887.
6. Bircher: Centralbl. für Chirurgie, 1893, No. 22.
7. Voss: Centralbl. für Chirurgie, 1902, No. 47.
8. Passow: Monats. für Ohrenheilkunde, 1912, p. 59.
9. Lombard, E.:   Annales des Maladies de l'oreille, 1904, Vol. I, p. 146.
10. Poulsen: Archiv. für klinische Chirurgie, 1896, Vol. 52, p. 460.
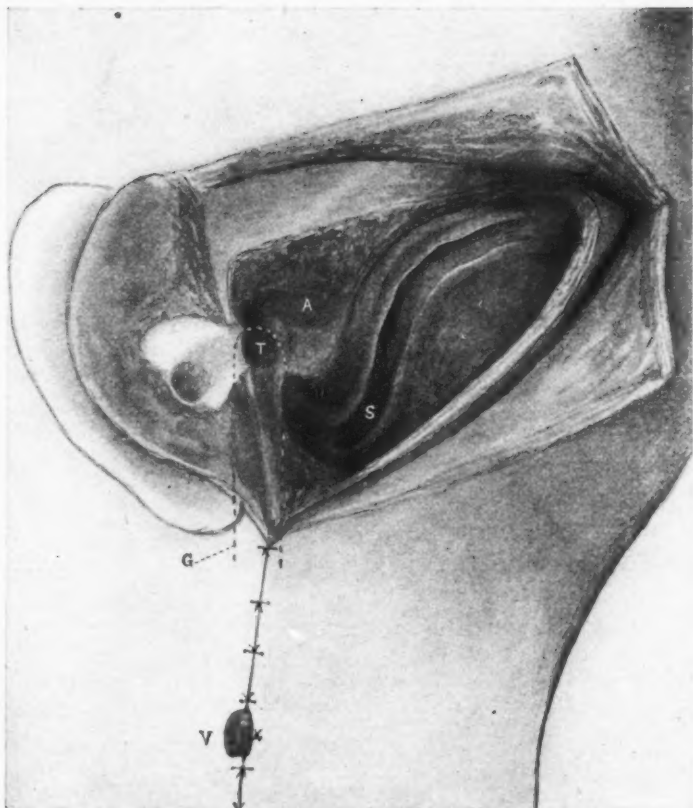
FIGURE I.

Sketch of First Operation, April 29, 1912.

The complete mastoid operation was done and the sloughed outer wall of the sigmoid was taken away for some distance, S. The jugular vein was divided and the end of the upper segment fixed to the skin of the neck, V. A, inner wall of antrum; T, inner wall of tympanum—posteriorly is seen the bend of the facial canal; G, dotted line shows course of jugular vein in upper part of neck. By working behind and internal to the descending part of the facial canal, the jugular bulb was in part exposed: the facial canal was first defined so as to protect it from injury. The bulb rose to a high position on the inner side of the tympanum.
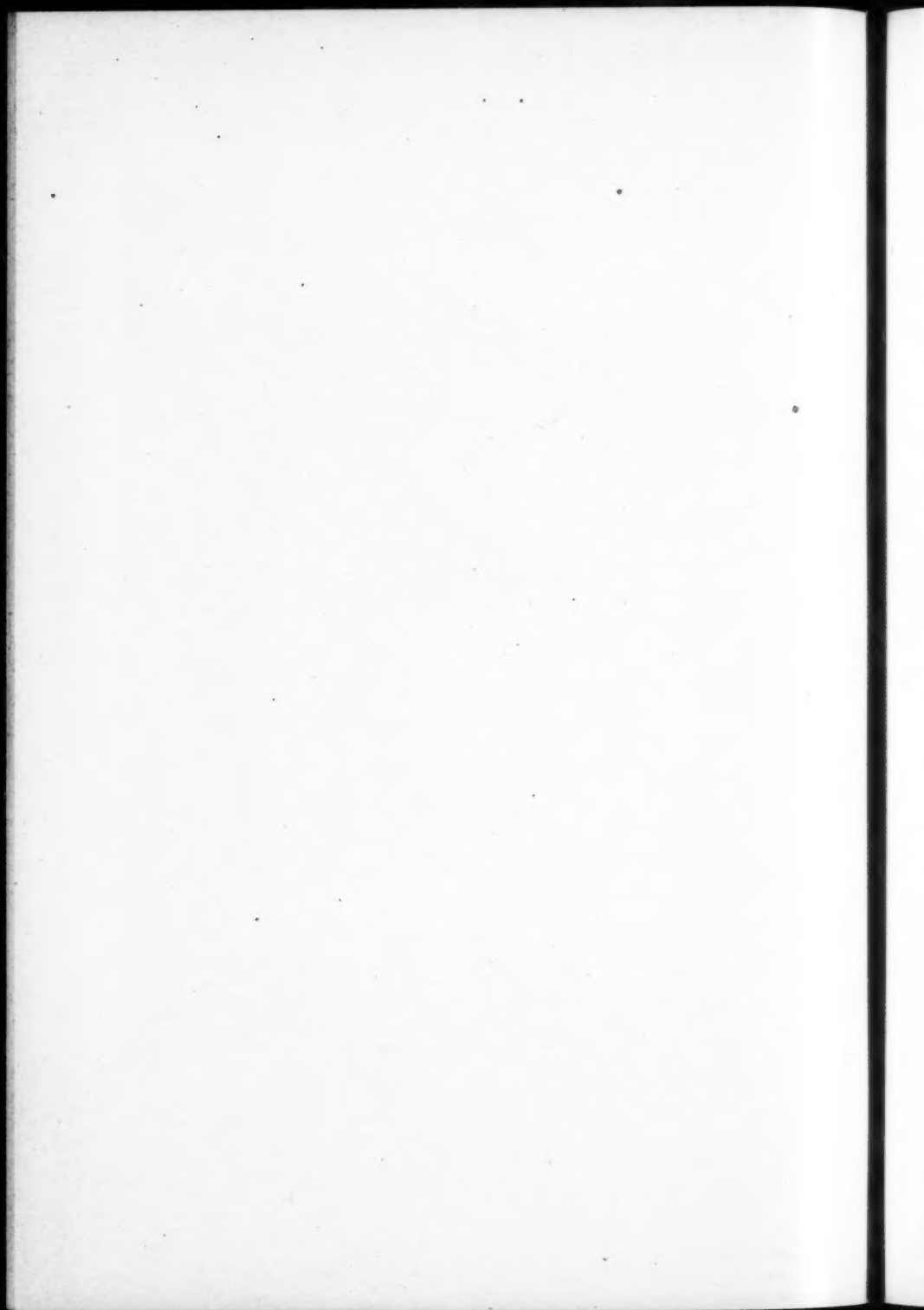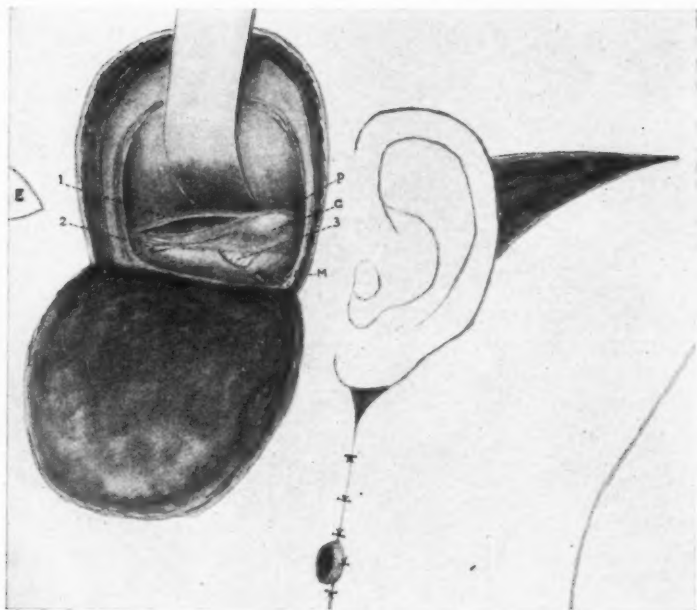
FIGURE II.

Sketch of Second Operation, May 8, 1912.   Exposure and Incision of
Cavernous Sinus.

1, First division of fifth nerve in outer wall of sinus; 2, Second division
of fifth nerve entering foramen rotundum; 3, Third division of fifth nerve
entering foramen ovale; G, Gasserian ganglion in cavum Meckelii; P, Su-
perior petrosal sinus; M, Middle meningeal artery tied above for foramen
spinosum; E, Outer angle of orbit.   Between the first and second divisions
of the fifth nerve an incision has been made into the cavernous sinus; out
of this incision came blood clot and pus.   The cavum Meckelii should not
be opened; if it is, the subarachnoid space is exposed to infection.
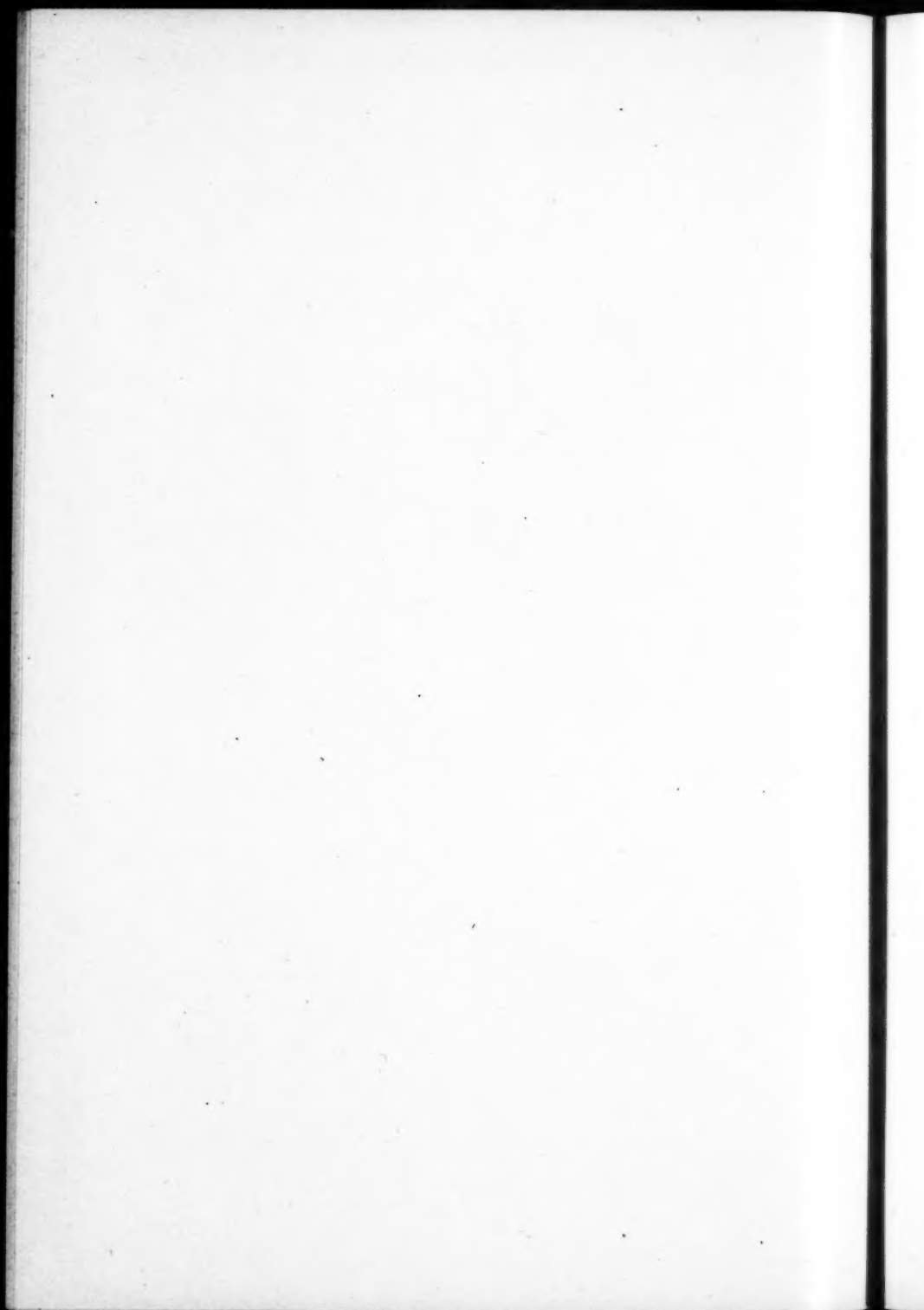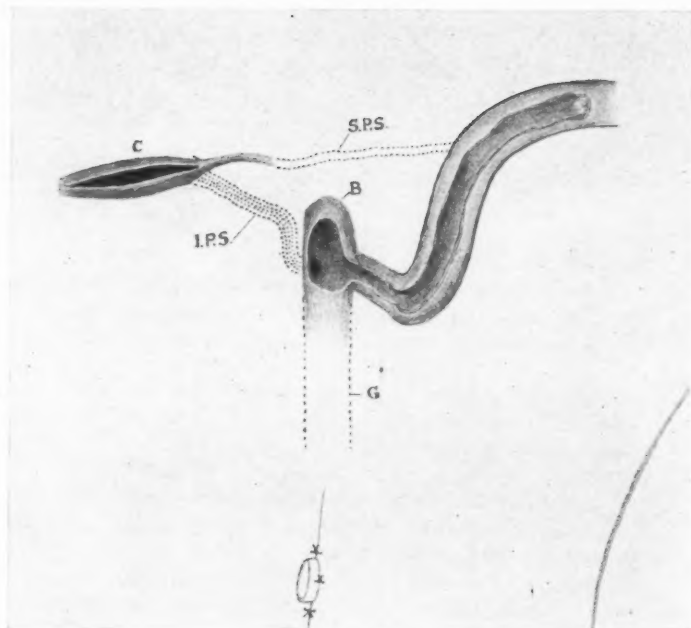
FIGURE III.

Sketch of the Venous Sinuses Involved in This Case.

The sigmoid sinus was opened up as far as the bulb in the first operation. C, The cavernous sinus (not affected). G, Internal jugular vein. I. P. S., Inferior petrosal sinus found full of pus at autopsy. I suggest that when the cavernous sinus has been infected by extension of the disease from the sigmoid sinus and bulb, that the bulb should be fully exposed and its wall cut away as in the above drawing, B. The opening of the inferior petrosal sinus into the bulb could then be identified, and by means of a syringe and small rubber tube the infection material in it could be washed from the bulb and out through the incision in the cavernous sinus.

LIX.

THE CONSERVATIVE TREATMENT OF CHRONIC AURAL SUPPURATION.*

By Sidney Yankauer, M. D.,

New York.

In the treatment of any disease the highest degree of perfection is reached when we have attained the complete restoration of the diseased parts to a perfectly normal condition. In the disease under consideration this means not only that the cause and the products of the suppurative process have been entirely removed, but also that the drum membrane has been closed and the hearing restored to normal. An attempt to achieve this ideal result has been made in the socalled conservative radical operations of Heath, Bondy, Siebenmann, and others. If the excellent results reported by these authors could be obtained in the majority of cases, and by most other operators, their methods would deserve universal acceptation; but the profound pathologic changes which take place after prolonged suppuration preclude the possibility of success by these methods in many, if not in most cases. In the radical operation as performed today by nearly all operators, the drum membrane and the ossicles are removed; hence the restoration of the tympanic cavity to a normal status is entirely excluded, and the immediate object of the operative technic is the removal of the cause and the products of the suppurative process. Now, it is a well recognized fact that the healing of any wound is not a direct result of the operative procedure, but that it is brought about by reason of the reparative powers of nature, which are physiologically inherent in all living tissues. A consideration of general surgical pathology teaches us that the surgical intervention initiates the reparative process by the removal of the causative factors

---

*Read before the Ninth International Otological Congress, at Boston, August 12-17, 1912.

(bacteria) and the products of the disease (dead tissue and secretions), leaving a wound surrounded on all sides by healthy tissues. Inasmuch, however, as the healing of the wound, especially a bone wound, takes time, it is necessary to sustain the reparative process, not only by rendering the removal of the disease products continuous and permanent, through the establishment of perfect drainage, but also to safeguard the wound with certainty against the return of the infectious agencies. In bone wounds in other parts of the body drainage can be established by operation, and reinfection can be prevented by proper surgical dressings. In the ear these measures do not suffice, as the wound cavity is in constant communication with the nasopharynx through the eustachian tube. That the closure of the eustachian tube is necessary in order to secure healing of the radical operation wound in the largest percentage of cases, is attested by the experience of most operators.

It is a familiar fact that spontaneous healing of chronic middle ear suppuration occurs in a considerable number of cases. The fact that such cases may have been of many years' duration, that the mastoid bone has become sclerosed, and that the ossicles may have disappeared, indicates that these cases were not merely socalled "mucous membrane" cases, but that the bone was also involved.

In order that spontaneous healing of the diseased middle ear and mastoid cells may occur, it is essential that the same conditions prevail which we seek to establish by our operative procedure, i. e., a passage for the escape of pus sufficiently free to prevent retention. When there is retention of pus in the middle ear or mastoid cells, nothing less than radical surgical intervention can bring about a cure. But retention of pus makes itself evident by symptoms; it is a rare case indeed in which the retention of pus is not indicated by the presence of either pain, tenderness or fever, labyrinthine or intracranial symptoms. In every case, therefore, in which these symptoms are absent, and have been absent for a long time, we can logically conclude that the drainage from the middle ear and mastoid cells is sufficiently good to prevent retention of pus, and that spontaneous or natural healing must eventually take place if we prevent reinfection of the ear through the eustachian tube. By studying the process of spontaneous healing,

therefore, we obtain the indications for the measures to be employed for the conservative treatment of the disease. These are, first, measures intended to prevent reinfection of the ear from the nasopharynx, and second, measures intended to aid in the removal of the secretion from the mastoid cells, the middle ear and the external canal.

Of the measures intended to prevent reinfection through the eustachian tube, the first to be considered is the removal of the source of the infection in the nose and nasopharynx. The frequent association of middle ear suppuration with nasal accessory sinus diseases, ozena, etc., is a familiar fact, and the necessity of the treatment of these conditions need not be dwelt upon before this audience. More worthy of consideration is the importance of the thorough removal of adenoids and tonsils, and the treatment of catarrhal conditions of the nose and nasopharynx following diphtheria and other infectious diseases in children. For even though children are more subject to the destructive inflammations accompanying the infectious diseases of childhood, nevertheless chronic aural suppuration is of shorter duration in children than it is apt to be in adults, and the reconstructive powers of the child's tissues are so much greater than those of the adult, that a complete restitution to a normal condition is much more likely to occur.

A second method of preventing reinfection of the ear is to neutralize the infectious agent by means of autogenous vaccines. Many glowing accounts of the action of vaccines on the ear have been published, but most of the work has been done by pathologists, not otologists, and an exact description of the pathologic changes in the middle ear before and after the treatment is lacking from many of the reports. There is no doubt that when the vaccines act, there is a marked diminution in the amount of the discharge, and sometimes complete cessation. When the preparation of these substances is better understood, much is to be hoped for from their use in this as in other suppurative processes.

By destroying or neutralizing the infectious agents in the nose and nasopharynx, a state of cleanliness is brought about in the middle ear, which, by persistent attention, may be maintained long enough to permit the ulcerated mucous membrane in the middle ear and mastoid cells to heal. But even if the local process in the middle ear has completely healed, the

patient is not permanently free from discharge or moisture in
the ear, as long as the eustachian tube is open and there is a
perforation in the drum membrane. Most patients admit that
from time to time there is some moisture in the ear, especially
after contracting a fresh cold in the head. When the drum
membrane is closed, it is difficult and usually impossible to
propel fluids through the eustachian tube into the middle ear.
This fact has been proven by experiment, and when it is de-
sired to inject fluids into the middle ear for therapeutic pur-
poses it is necessary to use the intratympanic catheter, which
must be passed beyond the isthmus to prevent the fluid from
returning into the nasopharynx. On the other hand, when the
drum membrane is perforated, fluids can be driven through
the tube with little difficulty. An individual who has a perfor-
ated drum and a patent tube invariably propels the secretions
of the nasopharynx through the tube into the middle ear every
time he blows his nose, and often when he coughs or sneezes.
The passage of air and fluids is accompanied by a hissing or
bubbling sound, but as this condition has existed for a long
time he may be unconscious of the fact, unless his attention is
called to it. When the tube has been closed by operation, the
patient recognizes the difference, for he has learned to realize
that he is no longer blowing his nose through his ears.

The closure of the eustachian tube by curetting or reaming
out its tympanic orifice is a procedure which has been prac-
ticed by otologists as a part of the radical operation for some
years; but it was not known that it was possible to reach the
isthmus of the tube through the intact external auditory canal,
and, therefore, without performing the radical operation, until
the writer published in the *Laryngoscope* in July, 1910, the
special instruments which he has devised for this purpose after
careful anatomic and clinical studies. The instruments and the
operative technic were fully described at that time and need
not be repeated here.

The immediate effect of the curettement of the tube is the
removal of its mucous membrane. It is not until granulation
tissue has formed and cicatrized that organic atresia can be
said to have taken place, a process which requires several
weeks. The tube is not always closed after the first curette-
ment; a second, and even a third curettement may be required.
The failure of the tube to close is due to three causes:

First. Insufficient curettement. When the tube is large and round, so that the largest curette can be used, the mucous membrane may be inverted like the finger of a glove, the cut end becoming visible in the ear. If I have created the impression in the article above mentioned that this occurred in most instances, I wish to correct that impression now; for a successful inversion is accomplished in a minority of the cases. This results from the anatomic formation of the tube, for examination of the skull will show that the cross section of the bony tube is usually an irregular triangle, with angles which are sometimes acute and narrow, into which the head of the curette will not enter. The shape of the isthmus may be determined before operation by observing the shape of the cotton tipped applicator with which the isthmus has been anesthetized, for the cotton tuft will assume the shape of the isthmus, and retain this shape for some minutes after its removal. It may also be determined by the use of the probe, and the experienced operator will recognize the presence of irregularities in the tube by the sensation imparted to the finger while using the curette. Under these circumstances the mucous membrane cannot be inverted in one piece, but is brought out into the middle ear in the form of shreds and small pieces. When its tube is triangular, curetting is completed by the use of smallest curette, for if even a fine strand of mucous membrane is left in tube, closure will not occur.

Second. The presence of adhesions near the tympanic orifice of the tube, which partly close off the tube from the middle ear. These adhesions will be referred to again.

Third. Suppuration within the canal for the tensor tympani muscle. This canal is separated from the tube proper by a thin bony wall, whose tympanic end is known as the processus cochleariformis. When this bony wall is complete the tensor canal ends near the isthmus as a blind pouch, and the canal is entirely separated from the tube. Dehiscences are frequently present in this wall, so that when the mucous membrane is removed from the tube, communication is established between the two canals. Not infrequently the canal is open at its medial end, so that the belly of the tensor tympani lies directly under the mucous membrane of the isthmus, some of its fibers taking their origin from the cartilaginous portion of the tube. When the isthmus is curetted in the presence of

these anatomic conditions, the tube itself may be closed, but a communication is established between the cartilaginous tube and the middle ear through the tensor canal. Under these circumstances, while the smallest filiform bougie cannot be passed through the tube into the ear, air can be blown through, and by filling the external auditory canal with water, the air can be seen bubbling through the water. When this condition is present, the processus cochleariformis must be broken down, so as to unite the two canals into one. After the removal of the anterior half of the outer attic wall, this can be readily accomplished by means of the same curettes which are used to close the tube.

After organic atresia of the eustachian tube has been established, the ear has become permanently separated from the nasopharynx, and is therefore entirely independent of any changes, acute or chronic, which may take place in the nose or nasopharynx. This statement is not based upon theory, but is the result of observation, for among the patients whose tubes I have closed, there have been cases of acute rhinitis, acute suppuration of the accessory sinuses, ozena, chronic ethmoiditis with the formation of polypi sufficiently large to cause complete nasal obstruction and mouth breathing. In all of these cases the aural suppuration had ceased after closing the tube, and the ears have remained dry in spite of the diseases which affected the nose, the oldest case under continued observation being of four years' duration.

The percentage of cases of middle ear suppuration which can be cured by closing the eustachian tube, and the length of time required for the parts to heal, depends upon the condition of the drainage from the mastoid cells and middle ear. When the extent of the diseased tissues in the ear is small, so that the disease products are scanty, and the passage for the escape of the secretions is sufficiently free, an early cessation of the discharge is to be expected. When the destruction of tissue is more extensive, the disease products more abundant, and the passage for their escape obstructed, the cure of the disease will be delayed, or may not take place at all. In the writer's cases, the period of time which has elapsed until the ear became dry has varied from a few weeks to eighteen months, the average time being six months. The total percentage of cures resulting from the closure of the tube alone has been

60 per cent, the oldest case having remained well for more than four years. In making up statistics on this subject, cases in which the tube has remained open should be excluded. For the number of cases in which a second or third curettage was necessary are of interest only in determining the technical value of the operative procedure. If the statistics are intended to determine the influence of closing the tube upon aural suppuration, only those cases should be included in which organic atresia has been actually accomplished.

In an abscess in the soft parts of the body, drainage is facilitated by the collapse of the walls of the cavity, but in the mastoid the walls are solid and cannot collapse. Moreover, the middle ear and mastoid cells constitute a very irregular and complicated series of spaces; hence, the drainage of these spaces is not a simple matter of gravitation, but is dependent upon the same physical laws which govern the drainage of the accessory cavities of the nose.

The most important of these factors is the powerful adhesion between the secretion and the surface upon which it lies. On account of the capillary attraction caused by this adhesive power, the secretion is enabled to creep along the interior surface of mastoid cells until it reaches the aditus and antrum and flows down into the middle ear. The fact that the middle ear is full of secretion does not indicate that the mastoid cells are full and overflowing; because most of the secretion found in the middle ear comes from the nasopharynx, having been blown into the ear through the eustachian tube. When the tube has been closed, the amount of secretion in the ear is always very much lessened. If, however, there is enough secretion to completely fill the tympanic cavity, further drainage from the mastoid cells ceases at once. For the surface tension of the secretion in the middle ear offers sufficient resistance to prevent further discharge from the mastoid cells by action of capillarity. In order, therefore, to aid nature in draining the mastoid cells, it is essential that the secretion be removed from the middle ear as fast as it accumulates therein. The removal of the secretion may be accomplished by dry wiping or by irrigations. As the viscid secretions are not readily absorbed by cotton applicators, dry wiping is effectual only in the hands of the physician who works under direct inspection. When the secretion is abundant it must be re-

moved from the ear at frequent intervals, by the patient himself, by means of irrigations. Objection is sometimes made to irrigations because it is said that the epithelium of the middle ear and canal walls become softened and swollen. This objection, however, is not in accord with the facts, because when the ear is filled with pus the epithelium is already saturated with moisture. It is essential that the ear should be thoroughly dried after each irrigation; for if water is left in the middle ear, the purpose of the irrigation is defeated, as the water offers as much obstruction to drainage as did the pus. By substituting water for the pus a decided advantage is gained. for the water is easily absorbed by inserting cotton tipped toothpicks or wicks of absorbent gauze into the ear, and the patient should be carefully instructed in the manner of using them.

The addition of antiseptics to the irrigating fluid probably serves no useful purposes, as the object of the irrigation is purely mechanical. Some antiseptics are objectionable because they coagulate the pus and interfere with its removal. This is true of all acid solutions, even of such a weak acid as boracic acid. On the other hand, alkalies, particularly borax solutions, have greater solvent power for mucus and pus than salt solutions, and when the secretion is thick and viscid some advantage is gained by their use.

When there is any obstruction to the outflow of the secretions, and this obstruction lies beyond the aditus ad antrum, i. e., within the mastoid bone, it can be removed only by opening the bone by some form of radical operation. If, however, the obstruction lies in the attic or middle ear, it can be recognized by inspection, and can be removed by minor surgical operations. As these operations can usually be performed painlessly under local anesthesia, and without keeping the patient in bed, they may be classed among the conservative measures, at least from the standpoint of the patient.

The drainage from the mastoid cells into the middle ear is totally free when the middle ear is open. By an open middle ear I mean an ear in which there is a large perforation of the drum membrane, and in which there are no adhesions or cicatricial contractures, especially between the remnant of the drum membrane and the inner tympanic wall. The absence of gross pathologic changes in the visible part of the middle

ear may be taken as indicating that the attic is similarly free, and that there is consequently no obstruction in the region of the aditus ad antrum. It is this class of cases which get well most rapidly after closing the eustachian tube.

When, on the other hand, there is evident caries of the ossicles, the swelling of their mucous membrane coverings is sufficiently marked to interfere with the flow of pus through the aditus. Hence, the removal of the ossicles is indicated.

Aural polypi frequently cause obstruction to drainage. As long as the eustachian tube is open, they recur rapidly after removal by the snare; when the tube has been closed, this tendency to recurrence disappears, and their surgical removal is necessary only when they are excessively large. Aural polypi are merely exuberant granulations, growing in the immediate neighborhood of carious bone, and they persist until the carious bone is removed, either by curettement or by the slower dissolving process of nature. When they are pedunculated in form, they can be shrunken by means of caustic applications; and when they have become so small that they are mere sessile masses, the growth of epithelium over them can be stimulated by applications of weak solutions of nitrate of silver of less than 2 per cent. The writer has tried many of the newer preparations for stimulating epithelialization, but has found them less efficient.

In the course of chronic suppuration adhesions are frequently formed in the middle ear and interfere with the drainage of the attic and mastoid cells. Sometimes these adhesions are caused by the fact that the edge of the perforation in the drum membrane is adherent to the inner tympanic wall, sometimes opposing masses of granulation tissue become united at their apices, and sometimes they are caused by contracture of the folds of mucous membrane which normally exist in the attic. In the upper part of the middle ear, such adhesions may extend from the processus cochleariformis backward nearly to the posterior wall of the attic. Above such an adhesion there is a pus pocket, which may be in communication with the eustachian tube through the tensor tympani canal.

Adhesions in the lower part of the middle ear cavity usually have the form of thin membranes tightly stretched between the annulus tympanicus and the inner tympanic wall. They are formed by the lower part of the drum membrane becoming

attached to the promontory. They begin at the upper border of the tympanic orifice of the tube, extend immediately above the hypotympanum to the posterior wall of the middle ear, forming a cover over the round window. They may be concave on their upper surface, and so be difficult to recognize without using the pneumatic speculum. Underneath such adhesions there is a fistulous tract, sometimes containing pus, which communicates with the eustachian tube in front, and which extends along the floor of the middle ear to the posterior wall, where it opens into the upper part of the tympanic cavity, or directly into the attic. When the eustachian applicator is passed through the tube, its end cannot be seen in the middle ear, but it causes the membranous adhesion to bulge inward in the form of a small prominence. In his article describing the condition which he calls tuborrhea, Urbantschitsch warns against the use of irrigations through the eustachian tube whenever a great deal of pressure is needed to force the irrigating fluid into the ear. The writer considers this advice well grounded, as he has found that it is only in cases where the adhesions just described are present, that much pressure is necessary to force the fluid through the tube. When these adhesions are present, it is impossible to pass any instrument into the tube for the purpose of curetting it, and when they extend posteriorly, they shut off the round window, and thereby cause a marked reduction in the hearing power.

These adhesions are cut with angular knives. As the membrane is usually tightly stretched, the lips of the incision retract, so that the fistulous tract underneath is exposed to view. It is now found that the eustachian applicator can be passed freely into the middle ear, that irrigations through the eustachian catheter can be carried out with a minimum of pressure, and the salpingian curette passes easily down to the isthmus. When all secretion has been removed from the niche of the round window, the hearing may be markedly improved.

Sometimes adhesions of a different type are found in the lower part of the tympanic cavity. They are formed of thick, swollen tissue, grayish and edematous in appearance. When incised the lips of the incision remain in contact, and it is difficult to recognize that there is a fistulous tract underneath them, as they are so swollen that the walls of the fistula are in contact. When the ear is inflated, either pus or air bubbles

appear in the incision. When suppuration ceases and the ear
has become dry, the point of attachment of such adhesions to
the promontory can be recognized as a thin white line.

From the above description it is evident that in order to ob-
tain satisfactory drainage from the mastoid cells (which is
absolutely necessary to secure healing of chronic aural sup-
puration by conservative measures), it has been necessary to
perform an intratympanic operation which included much
more than the removal of the ossicles. The term ossiculectomy,
therefore, does not properly describe it. In addition to os-
siculectomy, it was necessary to incise all adhesions, to remove
part of the outer attic wall, to break down the processus
cochleariformis, and to curette the eustachian tube from the
isthmus outward. The writer has performed such a complete
exenteration of the middle ear in a number of cases with suc-
cess, but the number is too small for statistical purposes.

Of all cases of chronic suppuration, the most difficult to cure
by conservative measures are the socalled attic suppurations,
with a small perforation in Schrapnell's membrane. When
caries of the ossicles exists in these cases, the auditory func-
tion of the drum membrane is destroyed, and the exenteration
of the middle ear is justifiable, and in the writer's hands has
been successful.

When organic atresia of the isthmus of the eustachian tube
has been successfully accomplished, certain important changes
of a trophic nature take place in the middle ear. Granulation
tissue shrinks rapidly and polypi lose their tendency to recur;
adhesions which have been cut do not grow together again;
the inner tympanic wall becomes pale, dry and leathery in ap-
pearance; and the perforation in the drum membrane never
becomes smaller, but, on the contrary, grows larger, so that
after a few months all that is left of the drum is a narrow
white band on the annulus tympanicus.

AURAL VERTIGO (NONSUPPURATIVE)—A CLINICAL AND THERAPEUTIC STUDY.

By Richard Lake, F. R. C. S., Eng.,

London.

For some years it has been very obvious to me that a definite clinical classification of cases of aural vertigo was most desirable. Much attention, as we all know, has been directed to the study of aural vertigo when complicating suppurative conditions. Much interesting work has been done in the endeavor to explain satisfactorily the mechanism, pathology, and histology of the semicircular canals and their influence on equilibrium. It is quite true that in England the number of cases operated upon for suppurative labyrinthitis has been remarkably small, in proportion to the number of ears operated upon for chronic suppurative disease, as compared with the Continent. Especially is this so when one considers the operative work of the large teaching schools of Germany and Austria.

Perhaps the primary reason for my desire to obtain a working clinical grouping or classification was more for personal than general use, but without the stimulus of an invitation, such as your chairman extended to me, it is probable that I should have been still waiting for somebody else to do the work. For when one commences such a classification, one sees only too clearly the limitations of one's personal horizon and the great difficulties which must be faced if one is to accomplish one's self-imposed task with any satisfaction. Equally evident it must be that the classification such as I am attempting is but an expression of personal views, wanting the light of kindly criticism combined with the enormous advantages to be derived from an aggregation of the views of intelligent and practical men. For it must have struck, and must strike, all of you how

hard it is for any writer on such a subject as aural surgery and therapeutics to place in a book new theories, as thereby he lays himself open to the very possible experience of finding that before his book has been long in the hands of his confrères, some fact has come to light which materially alters his views, and his book remains a standing monument of a good endeavor which has failed.

One of the chief difficulties that we deal with—in fact, I presume one may say the principal difficulty—is that all our reasoning about many pathologic conditions, especially aural vertigo and tinnitus, is based upon hypotheses, and these hypotheses being but the expression of the thought of an individual, such connot appeal to everybody. But after all, the human race is more content to accept than to speculate. Otherwise we would see much more marked progress in the elucidation of the abstruse problems of hearing and equilibrium than hitherto.

What I have said with regard to pathologic states being equally true of most of the normal physiology of the part, our difficulties are materially enhanced, and that is why I have chosen the title of clinical. For I believe that with our at present limited vista we must be content for the time being with careful clinical observation and careful clinical records, aided by deductive argument, until such time as may, and I trust will, come when definite, clear, and instructive pathologic evidence will be obtained which will shed so clear a ray of light upon our branch of the healing art, now shrouded in mystery, that those who live to see it will correct our speculations with their knowledge.

Vertigo only seems to have been considered as an aural symptom for a matter of some one hundred and sixty years, and even then more from a physiologic than from a pathologic and clinical standpoint. As far as I have been able to ascertain, by a somewhat cursory and casual investigation, none of the works upon aural surgery published in England until the middle of the nineteenth century had any specific allusion to vertigo; even Toynbee, that astute and far-seeing observer, is, as far as I can find, silent on this subject, and as an absolute matter of fact we must, I believe, look upon Menière as the first man ever known to give a definite account of a case of the kind, by a careful pathologic report of a case of aural vertigo, which aroused the interest of aurists and pointed out to them the

value of vertigo as a symptom. This symptom, however, has, as you know, taken many years to arrive at its present importance.

Vertigo, from an aural standpoint, became, and was for many years, only considered as Menière's disease. The result, of course, was confusion, and the lack of physiologic, clinical, and pathologic investigation forced that confusion to continue, which it did until the enormous stimulus to labyrinthine surgery was given by Jansen by his work on the labyrinth in suppurative disease. We began, as you know, in about 1904, to operate for intractable vertigo; and the very fact that one had commenced to operate, entailed a closer examination and investigation of those cases of vertigo which presented themselves for treatment. For the purposes of this paper I have taken the cases of vertigo which have come to my consulting-rooms during the last six years, some seventy in all, many having been sent to me solely for the purpose of deciding whether or not the case was a suitable one for operation. And I think, when I say that with two hospital appointments and my own private work the total number of cases on which I have operated for this condition is only fourteen, you will see that operation cannot be so very frequently demanded.

Of course, the large majority of the vertiginous cases occur as a complication of chronic progressive middle ear deafness. Equally true, a further fair proportion are caused by the effect of arteriosclerosis. The exact pathology, as I have before indicated, is quite unknown in many cases. What part is borne, for example, by changes taking place in the caliber of the aqueductus vestibuli, the canalis reuniens or semicircular canals, we are unable to say, but it has always seemed to me extremely probable that pathologic contraction of either of these tubes may have a distinct bearing on the question. I shall now, however, proceed to deal with these cases under the grouping which I present to you, not as a final solution of the clinical problems, but as a basis upon which to start.

AURAL VERTIGO.

Section I.—Peripheral causes. (a) Chronic progressive middle ear deafness; (b) hemorrhage and embolism into labyrinth; (c) traumatism.

Section II.—Aural vertigo due to altered state of blood

pressure: (a) Increased blood pressure; and (b) diminished blood pressure.

Section III.—Aural vertigo due to general systemic causes: (a) Leukemia; (b) occasional; (c) with ocular symptoms; (d) specific; (e) cerebral anemia.

SECTION I.

Group A. Vertigo as a symptom in chronic progressive middle ear deafness.—In other words, vertigo accompanied by a high-grade deafness occurring in patients usually below the age of forty. This arbitrary age limit enables us to eliminate the effect of arteriosclerosis. The group that we take first under consideration is one of very great importance, for it is within its confines that we find those cases in every way most suitable for operative relief, cases which are untouched by any other form of treatment. The typical cases may be divided into two classes: those which yield to treatment, and those which do not. There is no need of, or advantage to be derived from, detailing cases of aural vertigo occurring in chronic progressive middle ear disease, they are so common. When such a case presents itself one must investigate not only the patient's aural condition, but also carefully search for contributory factors capable of acting as the immediate stimulus to cause the vertigo, for in many of these cases stomachic or similar pathologic conditions may be contributory factors, and it is necessary that these be corrected before proceeding to treat the aural condition. When, however, there are no such contributory factors to be found, or when the case is clearly peripheral, one finds small doses of quinin combined with hydrobromic acid quite the most useful form of medication; though besides this, strychnin or large doses of valerian, combined with one of the mineral bases, are also efficacions. Electrical treatment is usually ineffective, though the effect of the high frequency current may certainly be tried.

When, however, one meets with patients in whom the vertigo is severe and often repeated, where the deafness is of a high grade, where the stapes is obviously fixed by osseous formation, where internal and external medication are unavailing, and where the patients' state is such that a continuation of life under such conditions is impossible by reason of their inability to earn their own livelihood, or some equally potent cause, then

one should without hesitation place before them the advisability of operation. In no case should operation be considered where the hearing is good, nor, I think, where it is useful. Where it is so reduced as to be a negligible quantity, the matter is different, and stands on the same footing as if the patient were completely deaf. For obvious reasons one finds these patients usually amongst the lower social grades. Thus, out of fourteen operations which I have performed, two were sailors, three clerks, three domestic servants, one seamstress, one hospital nurse, one medical man, one business man, and one a woman of independent means. The effect of the operation is certain. The relief is immediate and lasting, and the danger nil, of course provided that the minutest care and attention to detail is observed. And at this point I would wish to emphasize my strong adherence to antiseptic surgery, in contradistinction to aseptic surgery. First, I can see no advantage that aseptic surgery has over antiseptic surgery in this operation; secondly, as the great bulk of our aural surgery pertains to septic lesions, our staff, both medical and nursing, are more cognizant, at any rate in England, with antiseptic surgery; and finally—and this I take to be the most important point of all— it is, in my opinion, almost, if not quite, impossible to cut off the wound cavity from risk of infection, this risk being due to the existence of the eustachian tube, often abnormally free in these cases. I think that fourteen consecutive operations showing no mortality is not the least argument in favor of my contention. I purposely mention this, being a point that deserves attention and consideration.

Group B. Labyrinthine hemorrhage and embolism.—Although these two forms occur under widely separated conditions, it appears to me, for the sake of simplicity, to group them together rather than to separate them, thereby lessening the number of subdivisions. Hemorrhage takes place in patients beyond the prime of life, and is a direct result of arteriosclerosis, and is sudden in its onset. The chief evidence which one finds to corroborate the diagnosis of peripheral lesion lies in the use of the rotation test, aided by the caloric, which will show a diminished irritability of the labyrinth—that is, if the former test be employed fairly soon after the lesion has occurred, and before compensatory nystagmus has time to be established. It is also to be proven by the presence of a small

island or islands of tuning-fork perception still remaining in the cochlea, a condition which is seen also in traumatic affections of the labyrinth. The following is a case in point:

The patient was a man, aged fifty-five years, previously in good health. He was seen in September, 1911, and said that five weeks previously he got up one morning in his usual state of health, but while stretching himself "something went bang in his ear," immediately followed by right-sided deafness accompanied by vertigo and sickness, though the deafness was not noticed for twenty-four hours—that is to say, at the time the sickness ceased. The vertigo itself lasted two or three days, and he still has at times a sense of loss of coordination. The rotation tests and caloric tests showed a loss of irritability on the right side, while he was able to perceive the tuning forks between 64 and 256 double vibrations. The fact that deafness was not noted for twenty-four hours may either have been due to his being very unwell from the sickness, or on account of the blood having taken some time to find its way into the cochlea, but I think the former is more probable.

As a subdivision of this condition, we have cases of spontaneous vertigo which occur in quite young people, ages at which necessarily one cannot expect any arterial disease, and sometimes absolutely unassociated with mumps or any specific fever. In some of these one can only grope blindly for the causation, but when the patient is suffering from mumps at the time of the commencement of the vertigo, one is certain that an embolus has occurred, and, as in the third case, one is inclined to suspect that an embolus may occur in the labyrinth from septic conditions present even as far away as the other ear, but not proceed to endolabyrinthine suppuration.

1. A boy, aged sixteen years, felt giddy on rising one morning. The floor seemed to go up and down, and he noticed that he was deaf in the left ear. There was no tinnitus. Three weeks afterwards, on examination, one found that he was totally deaf on the left side.

2. A female, aged twenty-eight years. During a mild attack of mumps, one year previously, she awoke with vertigo, left-sided deafness, and tinnitus, the latter symptom persisting. There was feeble but distinct bone conduction, and complete aerial deafness. The rotation test in this case showed no ver-

tigo, but normal length of nystagmus, and the caloric test was normal on both sides.

3. A female, aged twenty-eight years, gave the following history: She had left-sided aural suppuration as the result of an acute otitis occurring during scarlet fever at the age of ten. At the age of fourteen she woke up deaf in the right ear one morning, with vertigo and vomiting, and rushing tinnitus, which lasted ever since. She was confined to her bed for two or three weeks with vertigo and sickness. On examination, one found that bone conduction still persisted, and that she could hear the tuning fork 512, 1024, and 2048 double vibrations.

These four cases are all similar, and yet dissimilar; in all there is distinct evidence of a peripheral form of lesion. In all one prominent symptom is noticed, not peculiar to this class, but, as we mentioned before, practically always noticed in otosclerosis—that is, the attacks always occurred in the early morning, the same reason doubtless occurring, except in the case of hemorrhage. That is, in the cases under the latter category, where embolism is the lesion, the heart action and the vital functions are at their lowest during the twenty-four hours, which would scarcely be the exciting cause in the case of a hemorrhage.

With regard to the therapeutic aspect of these cases, one must consider at once that cases of hemorrhage are absolutely beyond our reach. In all the other cases hypodermic exhibition of pilocarpin should be tried, if the patient is seen soon after the recurrence of vertigo—unless the use of pilocarpin is prohibited by the general state of the patient. But if one employs pilocarpin, it is my opinion that the size of the dose should be increased as rapidly as is consistent with safety, and that it should be persisted in for about two weeks. If one bears in mind the dangers likely to occur from its administration, and by means of other therapeutic agents one counteracts those dangers, and at the same time does not interfere with its action, one can make these doses much larger than would otherwise be the case. It has occurred to me only once to see a patient sufficiently early to be able to employ this remedy, and that was in a case which followed or occurred during an attack of influenza, in which the drug employed in the way I have suggested seemed to produce a very beneficial effect.

Group C. Traumatic aural vertigo.—Not infrequently one finds that in fracture of the base of the skull vertigo is complained of, though frequently as a transitory symptom. It is rare that this symptom lasts for more than six months, and then frequently when the patient turns his head away from the affected side, though inability to walk in the dark may last for some years. This symptom of inability to walk in the dark must, I believe, be due to a neuritis of the vestibular nerve, for it is found under two other pathologic states of the labyrinth, one that of chronic suppuration, and the other occasionally after an operation for labyrinthectomy. In the latter event it would seem possible that the destruction of the vestibular filaments has not been complete.

As an example of this a man, aged fifty years, was knocked down in the street by a motor car, and suffered from fractured base with hemorrhage from both ears. He was rendered completely deaf by the accident, and whenever he was moved in bed he was very giddy. When seen two years later, if he put his feet together and shut his eyes, he fell, usually towards his right front. Nystagmus could be elicited, more marked in the right side, by fixing the eyes on the finger and moving it rapidly from side to side. Both the caloric and rotation tests were negative. He was able to hear, or rather perceive, C, 512, 1024, and 2048 on the right side. Here again one notices the small island of the cochlea remaining that was capable of stimulation. Vertigo is also a symptom occasionally in rupture of the tympanic membrane; it is due, no doubt, to the forcible action of the compressed air upon the oval window or footplate of the stapes.

As another occasional cause of vertigo one finds the severe pressure of impacted cerumen upon the tympanic membrane and malleus, and still another cause is that of vigorous and persistent nose-blowing. But it is a condition which one can frequently meet with, especially in subjects of nasal obstruction, and not only is this a condition which we meet with not infrequently, but it is doubtless far more common than one thinks. For most people, on finding so obvious a connection between cause and effect, carefully avoid a repetition of so unpleasant an occurrence. What undoubtedly happens is that the violent inflation drives in with a sudden shock the footplate of the stapes, with the consequence that either the whole labyrinthine

fluid undergoes a momentary increase in pressure—a phrase, by the way, one must be most careful of using—or a wave is suddenly started which stimulates the hair cells, and one would never find vertigo as a symptom in instances where the tympanic membrane has stretched and become flaccid.

SECTION II.

Group A. Vertigo caused by increased blood pressure.— Arteriosclerosis often exhibits its first symptom or symptoms in the internal ear, and for this reason one frequently finds cases in which vertigo, as well as other aural symptoms, will lead to the diagnosis of arteriosclerosis in the patient. It will make its presence felt according to the age of the patient, not necessarily the actual age, but the age relative to the vigor of the patient, the activity of his mind and body, and the amount of work performed, whether corporeal or mental. One must in the majority of cases, at least, in which arteriosclerosis is complicated with vertigo, assume that the lesion is peripheral. This is actually so, I believe. In early cases all the signs and symptoms point in this direction, and they remain so, so long as there is increased blood pressure. Also, we know that, especially in those cases which start in relatively early adult life, there is a natural tendency for these most distressing symptoms eventually to disappear. This disappearance may be coupled with the loss of the hearing power in the affected ear. So in arteriosclerotic vertiginous patients we must consider each case on its own merits, and we must not indulge too freely in inductive argument. For whilst, as I have said, cases do proceed in the way described, the great number do not exhibit that tendency. Also we cannot legislate for a class of patients whose ages vary from forty years to the very extreme of life by any single rule.

Again, when we are considering, as we must consider, the question of treatment, especially when dealing with operative treatment, it is not only the problems of the disease, but the station, mode of life, and the importance of the wage-earning power of the individual that weighs down the scale. We must take into consideration also the patient's actual age, and his apparent age, his blood pressure, and his reactions to the various therapeutic agents to whose influence we submit him. We must not entirely omit in such a consideration even such obvious

points as his habits, the condition of his digestive organs, of his teeth, of his bowels, and of his kidneys. But, again, we must not allow any apparent cause for the trouble to be considered the cause, unless we are able fully to convince ourselves of its importance. Especially as regards the age, it has appeared to me from what I have seen that it is not advisable to speculate upon the probability of the disappearance of the vertigo by the gradual destruction of the vestibular nerve ending, as that is at the best a long process. This applies less, as already noted, in the earlier periods at which arteriosclerosis may affect the vestibular nerve, than it does when the patient has reached a more mature age, that again resolving itself into a question of the importance of the possession of stable equilibrium to the patient. For the younger the patient, in most instances, the greater the necessity for relatively rapid relief, though operation of itself should present no inherent veto, however old the patient.

Arteriosclerosis, however, presents in later adult life and at the commencement of old age another aspect of considerable importance, and that is that the vessels are now more universally affected, and so it is not always easy to determine with accuracy as to whether or not the disease is entirely peripheral, or it would be better to say as to whether the symptom of vertigo is due chiefly to a central or peripheral cause. Out of seventy cases, over twenty come under this category, though I am not proposing to give you the numbers of the cases which come under each of my headings, chiefly for the reason that with so small a number the permissible error of proportions is so enormous as to render such figures quite useless. When I referred to the central origin of vertigo as a symptom, I referred, of course, to a deficient supply of nourishment being supplied to the vestibular nuclei.

All these cases suffer with deafness in varying degree, and most of them one would find it hard to classify under any other group except that of the chronic progressive middle ear disease, but I believe the vertigo in all these cases to be distinctly due to the effect of arteriosclerosis. As examples of the gradual subsidence of the symptom, with its eventual disappearance, I will quote you two cases.

1. A male, aged forty-three years, a stockbroker. First visit, January 1, 1908. Slight deafness since January, 1907.

Some tinnitus. First attack of vertigo and sickness put right by blue pill (July, 1907). One bad attack, beginning in bed in morning. Still slight nystagmus to left. November 22nd two attacks, one on golf course and one after dinner (two have now been after dinner). January, 1908, another severe attack. Blood pressure, 170. December, 1909, no further attack.

The chief points of interest here were: The patient, a man with early arteriosclerosis, although quite young for that, living an extremely strenuous life, carrying always an enormous amount of speculative stock, and working more than hard in attempting to alleviate the conditions of life of those more humble and penurious. His bone conduction was reduced to but a small percentage, about one-fourth of the average. All seemed to me to point to a rapid loss of vitality in the terminal filaments of the right auditory and vestibular nerves. He was told to sell his stock, to go into the country, and to live a quiet, healthy life, with outdoor exercise. The patient in two years lost his vertigo, but he lost his hearing. Incidentally, he preferred this to operation and quick recovery.

2. A female, aged forty-six years. Seen March 20, 1908. History of tinnitus (right) commenced March. Vertigo one month—six attacks up to the time I saw patient. Seen again in October, 1908. Patient had had twenty-two attacks in interval, but tinnitus was better. By February, 1908, total attacks thirty-eight, when they suddenly ceased. December, 1909, caloric test (cold) on right side negative, and rotation test very much reduced. Bone conduction, C (128) = 38/25 inches. Rinné negative. Air conduction, C_2 to C_4.

In this case the patient undoubtedly suffered from arteriosclerosis, and the same result was obtained by waiting. It is to be remembered that in 1908 I had only performed the operation eight times, and although the results had all been satisfactory, the number was a small one, and I have no doubt the operation was not pressed. Also, neither of these patients needed to consider the problem of their livelihood; they were both of ample means.

The clinical aspect of an ordinary case of arteriosclerosis vertigo, I think, is much as follows: The special period for vertigo occurring is practically always found in the early morning. (This, however, as you will no doubt recall, is not abso-

lutely peculiar to arteriosclerosis.) It occurs in patients over the age of forty, but occasionally slightly younger, sometimes without any previous aural affection, but usually in a patient who is deaf to a greater or less extent. It is almost invariably accompanied by an increase in the deafness. The blood pressure upon examination will be always found to be raised. Occasionally there will be symptoms found which point to other organs being affected, notably the kidney. The attacks usually tend to diminish in violence, and still more frequently it will be found that if the patients are subjected to treatment with a view to lessening their arterial pressure and towards the checking of the disease of the arterioles, the tendency is for the patient to recover as far as the vertigo is concerned. The particular line of treatment which should be adopted I do not think is within the scope of our specialty. Personally, I invariably confine myself to the exhibition of three drugs—hydrobromic acid, with a small dose of quinin, or iodid of potassium. If these drugs fail to afford relief, I invariably advise the medical attendant of the patient that the patients' best interests are conserved by placing them under the care of a general physician. It is largely, if not entirely, due to Dr. Schaumberg that one has been able to speak with such great certainty on this point, for we all remember and appreciate his valuable work on the arterial supply of the inner ear. And it is, I take it, to the fact that the internal auditory artery is devoid of anastomosis that this organ is so easily affected in this way.

Group B. Vertigo occurring in patients suffering from aural disease who have a diminished blood pressure.—This group is an extremely interesting one. I only quote two cases again, as being fairly pathognomonic. One finds in the first case a hyperexcitable labyrinth to the caloric test, and in the second case diminished excitability. Both showed an extraordinary reaction on rotation, both becoming very markedly giddy, so much so that some period of time elapsed before they recovered, and in both the blood pressure was still further diminished by rotation. Although I have not given the exact figures, as the state of the patients made exact investigation impossible, yet in view of the excellent work done by Dr. Byrne of New York, it is interesting to mention the fact. And it is also interesting to notice that it is very rarely that one obtains exaggerated vertigo on rotation where the patients are suffer-

ing from increased blood pressure. But of one thing one may be quite certain, and that is these cases are not at all suitable for operation, unless one fails to relieve the patient by drugs and other appropriate treatment, given with a view to restoring the blood pressure to its normal. Nor even when one fails can operation always be considered, as in both these patients, for example, where the disease was bilateral, a diminished blood pressure, if at all marked, would, I think, militate against operation. Again, we are not, I think, prepared to say definitely that the effect is entirely due to peripheral trouble. Is it not equally probable that a want of nutrition to the central nuclei, at least, plays a part in producing this symptom of vertigo? I think we must answer this question in the affirmative.

With regard to the therapeutic aspect, the drug which has given me the best results is ernutin, and as you will see in the second case, it is only by the constant administration of that drug that the symptom is kept in abeyance.

1. A man, aged 55 years, a clerk, looking at least sixty-five to seventy, and certainly of a nervous temperament, came complaining of vertigo and deafness. The deafness, which was not very sereve, had existed twenty years; the giddiness had been noticed for the last few years. During the attacks the objects moved from left to right more frequently than in any other direction. The patient frequently staggered and could not walk straight. February 23 and March 5, 1912, rotation test, ten times in twenty seconds. Terrific vertigo, vomited once. On checking the chair he almost shot out, falling to one side in a state of collapse. Caloric test, irrigation with cold water gave rise to a similar severe attack. Blood pressure was abnormally low, only 105. It seemed quite probable that the low blood pressure was directly connected with the vertigo. In consequence the patient was given ernutin, ten grains three times daily, with the most satisfactory results. Since the commencement of the treatment no attack of vertigo has been recorded, and on April 1, 1912, he was reported as quite well.

2. A female, aged 49 years, of poor physique, was first seen on May 2, 1912. She had moderate bilateral deafness of eight years' duration and bilateral tinnitus. Paracusis Willisii and double incipient cataract. (Premature senility.) Vertigo for three years. Since her first attack she has had great difficulty in walking straight, staggers, and often almost falls. Each

time that she has one of these attacks, vertigo and nausea are well marked. She tends to fall to the right side, and objects appear to move from right to left. Turning the head usually produces an attack of intense vertigo, with movement of objects from right to left, and a tendency to fall to the side towards which the head was turned. This also is the case when the patient is lying down. In walking the patient must walk straight and not attempt to turn her head. She has only actually fallen once. She knows what may happen, and holds to something until the attack passes off. Rotation test, ten times in twenty seconds. Excessive reaction; intense vertigo; nystagmus. Blood pressure, after rotation, 95 clockwise, 105 counter clockwise. Caloric test, slight nystagmus only. Strychnin and atropin (Byrne) seemed to make the patient worse. She has now been on ernutin seven months, and is very much better. Whether she is well remains uncertain, but after she was at one time apparently well we found on stopping the administration of the drug that her trouble returned.

SECTION III.

Group A. Leukemia.—Menière's disease is, of course, the best known form of aural vertigo. It is most clearly defined and its pathology accurately known. For this reason I shall treat it briefly. It is only found in cases of leukemia or allied conditions, such as pernicious anemia. The patient is attacked with sudden deafness and vertigo. The deafness is immediate and complete, the vertigo transitory, though extremely severe. Should the patient be erect at the time of the occurrence of the lesion, he falls. Vomiting and sicknss are practically always present. Should the patient live for any length of time, as I have said, the symptom of vertigo disappears, but the deafness remains. The only case I have seen—which has been published elsewhere—is that of a woman who was suffering from leukemia, and presented all the symptoms above referred to. She lived for some six weeks after the hemorrhage into the labyrinth. Treatment here is quite useless and need not be considered.

Group B. Casual or occasional causes.—The most usual casual cause of aural vertigo I consider to be gout or gouty dyspepsia, which is also a frequent or casual cause of deaf-

ness and tinnitus. These cases are apparently aural, but they
have one peculiarity. At the time, or frequently some time
before the attack of vertigo, deafness and tinnitus are in-
creased and become pronounced. After the attack of vertigo
the deafness gradually disappears, and the hearing becomes
as it was before the attack. The following is a case in point:

The patient was a man of full habit, forty-six years of age,
who had suffered from slight right catarrhal otitis media. He
gradually became subject to severe attacks of vertigo, which
for some time I was at a loss to account for. I found that
for some short time before his attack of vertigo his deafness
was markedly increased, and that after his attack his deafness
lessened, and his hearing rapidly returned to what it had been
a few weeks previously. As I knew him to be of gouty habit,
and found that when his attacks of vertigo were commencing
he suffered extremely from flatulence, I sent him to a physi-
cian for general treatment, with the result that as his gouty
diathesis was got under control his attacks of vertigo ceased.

In such a case, of course, it is an open question as to
whether the condition was partly peripheral, and I am inclined
to think that his vertigo was not entirely due to his stomach
condition, as it frequently is in cases of vertigo occurring dur-
ing severe bilious attacks, where it is probably the result of
a much lowered blood pressure.

Group C. Cases in which vertigo is combined with ocular
symptoms.—These may be classed under two headings—the
first, that class described as aural epilepsy, in which the pa-
tients have a visual aura that gradually involves the auditory
vestibular nerve. These cases are not very common, at any
rate in our consulting rooms. I have only seen one case, in
which the patient suddenly had the most extraordinary optical
aura, seeing a sort of mirage, with a brightly colored sun and
waving fields, which was followed rapidly by intense vertigo
and sickness. These cases yield very readily to treatment;
small doses of quinin with hydrobromic acid seem to act as a
specific. The other class of case in which ocular symptoms
are combined with vertigo are most distinctly obscure, and
the following is an example: The patient, a male, fifty-four
years of age, had long resided in India, and returned to
England in 1906. On his arrival he contracted a severe cold,
which was followed by tinnitus. He had an operation in

1907, when his left kidney was removed. In the following year vertigo commenced, and ever since he has been subject to attacks of vertigo whenever he was subjected to a bright light. Also, curiously enough, loud noises are able to cause the same result. The left ear was markedly deaf, and he was examined carefully by great oculists, who have been unable to find any cause for the condition, and I must admit that I was in the same plight. The patient's very indifferent health would have precluded operation, if one had felt inclined to suggest it, of which I have some grave doubts. For the rest no treatment seemed to give him the slightest relief.

Group D. Specific.—No doubt if one were able to have the control of the examination of numbers of cases of nerve disease of specific origin, such as occurs in the large hospitals for diseases of the nervous system, one would come across many cases of vertigo, especially in tabes dorsalis. In tabes, vertiginous attacks occur combined with deafness and severe tinnitus in the form of a crisis. It is quite likely that they are as much due to the central lesion as to a neuritis of the auditory nerve, and as far as my experience goes, one notices in these patients other ocular symptoms of knee jerk, although the tabetic gait may not be noticeable. It is rarely that one finds vertigo as a symptom of aural specific disease unless there are at the same time symptoms of a graver lesion in the cord.

Inherited specific disease, more than the acquired, produces a form of nerve deafness frequently associated with vertigo, and in these cases, so far as my own experience goes, the vertigo is not a lasting symptom, but with the disappearance of the vertigo the hearing is destroyed. I am inclined to consider that in the acquired one finds a more hopeful prognosis than in the hereditary. But here, as in all forms of specific nerve lesions, treatment must be very prolonged, and a careful watch kept for any return, or threatening return, of the symptom, and it is well after the regular treatment has ceased that the patients submit to a six weeks' course every twelve months, using for choice either iodoform in five-grain pills or one of the modern iodin preparations.

Group E. Cases in which cerebral anemia simulates aural vertigo.—The group we now deal with is one which, strictly speaking, you perhaps will consider should not have been

introduced; but my reasons for introducing it are its value as a contrast, and that the group itself seems to me to be of interest; besides which, these patients are sent to us as aural cases, for in all of them deafness is present. These cases appear to me to be due to cerebral anemia affecting, at any rate principally, the deep nuclei of the eighth pair of nerves, though whether or not you will agree with my hypothesis, as I am afraid it is, I shall no doubt learn. These cases seem to show one sign in particular, which makes it extremely difficult for me to consider that these are due to a peripheral and not to a central disturbance, and that is a feeling of general surface warmth and of flushing, sometimes accompanied by sweating, which was present in the attacks, a condition which those of you who have suffered from mal-de-mer may possibly recall as a not infrequent concomitant, and a symptom also which I have not elicited in cases of obvious aural ver-·tigo. And I believe that the reason that we see these patients is because they are suffering from deafness, and the not unnatural assumption is that they are suffering from aural vertigo.

1. The first was a well-known physician, aged sixty-one years, with only one (the right) serviceable ear. He worked extremely hard, he traveled all over the country, often sleeping in trains, or getting but little sleep. He lectured on medicine, and of late had been extremely hard put to prepare a new series of lectures. The hearing in his right ear began to deteriorate, and this no doubt added its quotum to the overstrain. Easter, 1910, he went for a golfing holiday, forgetful of his age and of the age of his arteries, and played three rounds a day. A few mornings later he awoke with violent vertigo, retching and vomiting. His body felt very warm. The vomit was bilious, and the direction of objects left to right. He recovered completely. His blood pressure was 150. Again, in July of the same year the same series of events led to a second attack. They were always in the morning. Yet the patient did not take warning, and late one night he fell, seized with sudden vertigo. He sweated freely and vomited (note sea-sickness). He could not be moved for three hours. When I saw the gentleman in question he was still going ahead as if he were in the prime of life. He had occasionally a severe attack. He got quite over his attacks

by taking life less strenuously, and by the addition of a little alcohol to his dietary.

2. A female, aged sixty-two years. A great sufferer from rheumatoid arthritis, and almost complete deafness in the right ear. The patient, despite her severe handicap, was a most energetic and hard-working person, busy with good works. September 3, 1912. For about three years she had suffered with left eustachian obstruction, gradually becoming more and more deaf. On January 1, 1912, the patient had a severe attack of vertigo in bed in the morning, with objects rotating clockwise in a vertical plane. She has had four or five severe attacks since, and many mild ones. The attacks are heralded by flushing and by a feeling of surface warmth, and at times this is accompanied by perspiration. The patient has, however, had three attacks of vertigo without these symptoms. She could hear a whisper on the left side four inches, and C 32 to C 4096 double vibrations. Her blood pressure was 100.

RÉSUMÉ.

Chronic progressive middle ear deafness and arteriosclerosis are thus, according to my investigations, the most frequent causes of aural vertigo, and fortunately one finds that a large amount of benefit can be derived from the use of drugs in these cases, though by no means all of them are capable of this relief. We have also seen that operative interference is justified, and where it is used it should be uniformly successful.

There are two points which I expect will have struck you in my paper; one is the small reliance placed on the rotary and caloric reactions, and the other is the total omission of that socalled group in which increased labyrinthine pressure is said to exist. It is not, perhaps, quite within the scope of the paper to give at all at length my reasons for not employing the tests, nor for placing more reliance upon them. I am convinced in my own mind that one is able to make one's diagnoses and one's prognoses in the class of case under consideration equally well without their employment. I must, however, admit that, especially in cases of low arterial tension, the results were most extraordinarily interesting. With regard to increased intralabyrinthine pressure, although I have operated on at least twenty cases of labyrinthine vertigo where

there had been no perforation into the labyrinth—fourteen of
them where there had been no previous suppuration, and the
rest where there had—I have only twice seen any fluid on
opening the labyrinth.   In these two cases there was a con-
siderable amount of fluid; one of them also had an extremely
large external semicircular canal, apparently four or five times
greater than normal.   But even then I am not prepared to
say that these cases contained fluid under pressure, and I
have consequently thought it better, as the matter is treated
entirely clinically, to omit this possible factor.   But it is
rather difficult, indeed, to understand, from a purely mechan-
ical point of view, how fluid can be retained under pressure
in such a nonvascular bony cavity as the labyrinth.

The use of ernutin was suggested by my senior clin-
ical assistant, Mr. A. F. Penny, of London.   I found it
impossible, even in so lengthy an analysis, to contrast and
apply Dr. Kerrison's classification to that I adopted, so pre-
ferred to leave it, hoping at some future time to give it the
place it deserves and derive from it the benefits it possesses.
And, in conclusion, I must thank you again for your very flat-
tering invitation to me, and for the kind and sympathetic
attention with which you have listened to my lengthy paper.

BACTERIOLOGIC DIAGNOSTIC AIDS IN ACUTE OTITIC DISEASE AND ITS COMPLICATIONS.*

By Frederic E. Sondern, M. D.,

New York.

The frequent reference to the bacterial nature of the infection, in the discussions concerning diagnosis and prognosis of acute otitic disease and its complications, has naturally prompted the inquiry to what extent this information is of value to the clinician. If any rules can be formulated, in order to inspire confidence, they must have the broad base of practically uniform result obtained by a variety of observers under different environment and methods, rather than the narrower base of individual effort, no matter how efficient this may be. Many a competent worker with wide experience in his specialty has failed to see certain phases of a condition noted by another of possibly more limited activity. Such conditions are possibly unusual or exceptional, but by no means justify an assumption of inefficient methods or lack of proper observation. When an eminent worker in different environment reports bacteriologic results varying from our own, these possibilities must be incorporated in the broad rules established for the guidance of all, and an error in technic cannot be assumed to account for the difference in experience.

A knowledge of the nature of the original infection is undoubtedly of value, and the occurrence of an infection of the general blood stream also has diagnostic significance of no mean importance, but the broad experience of the clinician demonstrates that dogmatic rules concerning the meaning of one or other bacteriologic factor are bad. This is no attempt to belittle laboratory help in diagnosis and prognosis; quite the contrary. The best service from these aids is obtained with a full knowledge, not only of their value, but also of

---

*Read before the Ninth International Otological Congress, at Boston, August 12-17, 1912.

their limitations, and it is to a brief consideration of both that attention is invited.

The subject of bacteriologic diagnostic aids in acute otitic disease and its complications may be subdivided as follows:

1. Bacteriology of acute middle ear infection and its complications. (a) Types of infection and relative frequency of each. (b) Prognostic significance of the respective types.

2. Bacteriology of secondary infection of blood current following acute otitic disease. (a) With sinus thrombosis. (b) Without sinus thrombosis.

3. Value of blood culture in diagnosis. (a) Positive cultures. (b) Negative cultures.

4. Conclusions.

1. Bacteriology of acute middle ear infection and its complications.—Modern literature quotes the result of an exceedingly large number of investigations concerning this point, and while observers vary more or less in experience as to the relative frequency of one or other type of infection, the general opinion is fairly uniform. All bacteriologists in this field appreciate the difficulties of differential diagnosis in spread specimens made immediately after paracentesis, and the advantage of a culture made in addition to the spreads is obvious.

(a) Types of infection and relative frequency of each.— The order of relative frequency of infection is found to be as follows: Streptococcus, 50 per cent; staphylococcus aureus or albus, 14 per cent; pneumococcus, 14 per cent; streptococcus mucosus, 10 per cent; and the remaining 12 per cent showing no organism or one or other of the following: Proteus group, colon group, diphtheria bacilli, anerobic organisms and unidentified bacteria. Approximately 20 per cent of the specimens show mixed infections which seem least common with pneumococci and next with streptococci. These figures agree in the main with those of Libman[1] and others quoted by him, excepting a somewhat larger relative number of staphylococcus infections. This would seem explained by the fact that the figures were obtained in private and not hospital practice.

(b) Prognostic significance of the respective types.—Impartial consideration favors the view that, while a prognosis cannot be based on the nature of the infecting organism, some infections are more liable to produce complications than others. The streptococcus is certainly the most frequent invader

of the mastoid and deeper structures. The streptococcus mucosus and the staphylococcus are next in the order of frequency, the former, however, usually produces a far more virulent process. The pneumococcus, it was believed, rarely caused mastoid involvement and has never been seen in sinus thrombosis by some. With due regard to the difficulty in differentiating streptococci and pneumococci, this cannot be held to explain the publication of cases of pneumococcus bacteremia of otitic origin with and without sinus thrombosis.

While Netter, Jürgens, Leutert and others strongly maintain that the streptococcus is the only organism which causes pyemia, Hasslauer[2] mentions six cases of pneumococcus sinus thrombosis in a series of fifty-four cases, and one case of the Friedlander organism in the same series. He also quotes cases of Zaufal, Brieger, Condamnin, and Weichselbaum.

Urbantschitsch[3] also credits Brieger with the statement that pneumococcus bacteremia occurs as the result of otitic disease with and without sinus thrombosis, and that it gives rise to multiple suppurations in the various organs.

Dench,[4] Alexander,[5] Rolly[6] and Hoffman[7] also describe interesting cases.

It does not seem reasonable to suppose that the pneumococcus is less virulent in the ear than elsewhere, and there is ample evidence of its tendency to general systemic infections in the communications of Desguin,[8] Meyer,[9] Carr,[10] Adams[11] and Claude Sourdel.[12]

2. Bacteriology of secondary infection of blood current following acute otitic disease.—The most frequently encountered organism in the blood stream is the streptococcus; the next in order of frequency is the staphylococcus; third, the streptococcus mucosus, and finally the pneumococcus. In other words, the relative frequency of the different organisms in causing general systemic infection is nearly the same as that noted in the original extension of the inflammatory process. This relative frequency must not be confused with the relative severity or virulence of the resulting infection.

The virulence of the infection cannot be gauged by the organism any more than can the probability of extension, but in a general way it may be said that the streptococcus mucosus is the most virulent, with the other more frequent ones in the

following order: Streptococcus, staphylococcus, and pneumococcus.

The manner in which the infection gains access to the general circulation is still the subject of more or less controversy.

(a) General infection with sinus thrombosis.—It is generally recognized that certainly in the majority of instances a sinus thrombosis is the direct cause of the general systemic infection when this follows acute otitic disease. Otologists have mapped out a fairly clear clinical picture of this complication, and its prompt and accurate recognition is the rule rather than the exception. In the exceptional instances, however, where the clinical evidence is not typical and where there is a suspicion of intercurrent disease, not due to the otitic infection, the bacteriologic aids undoubtedly offer assistance. Unfortunately the claim that a bacteremia indicates thrombosis, and the absence of a bacteremia indicates the probable absence of involvement of the sinus, has too many exceptions to make it a practical working rule. Oppenheimer's[16] belief that a persistent streptococcemia in cases of chronic middle ear disease indicates an infected sinus thrombosis, quite agrees with personal experience and has the support of the majority of observers, but this does not hold good in acute otitic disease.

(b) General infection without sinus thrombosis.—Laboratory experience coupled with accurate clinical description offers ample evidence that, while sinus thrombosis is the most frequent manner of invasion of the general circulation, still, general infections do occur without sinus thrombosis, as shown by positive blood cultures and metastases without the characteristic symptoms of thrombosis of the sinus. In a number of cases of this kind, with local attention to the pyemic abscesses, close observation failed to show clinical evidence of sinus thrombosis, and with gradually decreasing bacteremia full recovery resulted without exploration of the sinus. On the whole, general infections without sinus thrombosis often end fatally, irrespective of the type of infection. While Leutert,[18] Jansen, Schencke, Heine and others maintain that a sinus thrombosis is the only origin of otitic pyemia, the experience detailed above is supported by accumulating evidence in recent literature that an infection of the general blood stream can occur without involvement of the sinus, and in this connection Koerner's description of osteophlebitic origin is note-

worthy. While such infection without sinus involvement is admittedly infrequent, it seems unreasonable to infer improper observation on the part of those describing it, as Jansen and others would have us believe.

Hoffman[14] reports a case of double mastoid involvement of meninges and pyemic abscesses without involvement of sinus or jugular bulb. Chmourlo[15] also opposes Leutert's theory with the statement that the two types of otitic pyemia are met with, and that the infection without sinus thrombosis occurs through the lymphatics. Urbantschitsch[3] also believes that a general infection of lymphatic or osteophlebitic origin may occur without sinus thrombosis, and supports Koerner in distinguishing between otitic pyemia with and without sinus phlebitis, claiming that sinus thrombosis is more apt to occur in the chronic cases. He describes in detail a general bacterial infection where death occurred nine days after the onset of the acute otitis with rapidly increasing staphylococcus bacteremia, persistently high temperature without chills or remissions and no metastatic foci. On autopsy, characteristic lesions were found in the heart, kidneys and retina, with perfectly normal sinuses. H. Luc,[17] Rimini,[18] Gilbert,[19] Zebrowski,[20] F. Müller,[21] Wild,[22] Kobrack[23] and others all describe cases which seem to distinctly demonstrate general infection without affection of the sinuses. Hayman[24] undertook extensive experimental work in the matter of sinus thrombosis and otogenic pyemia, and many of the results may be of distinct clinical value, as follows:

1. Experimentally, infectious sinus thromboses are best produced by way of the external wall of the sinus, through the application of an infected tampon.

2. The agents may reach the blood current without mediation of a thrombosis. The formation of a thrombus at the site of the infection is not absolutely necessary for the occurrence of an otogenic pyemia.

3. The thrombi may contain bacteria from the start, the agents as a rule having entered from the outside; but they may have been derived from the blood.

4. The contents of the thrombi in bacteria are variable; the ends as a rule are poorer in bacteria.

5. It is a noteworthy fact that the experimental infectious sinus thromboses show a pronounced tendency towards a spon-

taneous cure. The organization processes start at any early stage. At the end of about seventeen or eighteen days, originally infectious thrombi had become organized, or recanaliculized, although in some cases the external sinus wall had been completely destroyed by the infection.

6. From the macroscopic behavior of the sinus wall no positive conclusions can be drawn as to the sinus contents, nor does the macroscopic appearance of a thrombus afford any information as to its benign or malignant character.

7. Aseptic interventions upon the sinus do not lead to thrombosis.

8. In the presence of experimental bacteremia, aseptic interventions upon the sinus, which are otherwise tolerated without any harm, were observed to lead to extensive thrombosis.

He concludes that direct invasion of organisms into the blood current is certainly possible without sinus thrombosis, and adds that surgical intervention upon the normal sinus, if a bacteremia exists, will be followed by thrombosis which may seriously complicate the course of the experiment.

Thus the detailed personal experience seems amply supported by clinical and experimental data, and it would seem wrong to assume that infection of the general blood stream cannot occur without sinus thrombosis, though this is without doubt the most common mode of infection.

If sinus thrombosis is to be made a foregone conclusion in general infections of otitic origin, then logically a venous thrombosis of a large vessel near the point of entrance of any infection must be insisted on, and this is far from general experience. The exposure of the sinus and its direct connection with the infected wound is obviously not an indifferent procedure, and for this reason it is important to appreciate that a bacteremia or evidence of infection of the general blood current is not sufficient evidence alone to justify such operative procedure.

3. Value of blood cultures in diagnosis.—In cases of acute otitis without or with mastoid involvement, irrespective of the grade of toxemia, microorganisms are usually not present in the circulation, and blood cultures are sterile. In exceptional instances positive blood cultures are obtained, and owing to the subsequent clinical course and convalescence there is no valid reason to believe that an unrecognized thrombosis

of the sinus existed.   This experience is corroborated by Duel and Wright[25] and others.   As previously detailed, some of the most virulent and rapidly fatal cases of general infection following acute otitic disease occur without involvement of the lateral sinus or jugular bulb, and the bacteremia is accompanied by a train of symptoms quite different from that noted in sinus thrombosis, which should permit a differential diagnosis.

Cases are not uncommon in which an acute otitis develops during an acute infectious disease, such as scarlet fever for example.   In an instance of this kind a positive blood culture may be referable to such disease and not to an extension of the otitic infection to the general circulation via the sinus or other channel.   Bennecke[26] obtained positive blood cultures in 32 per cent of scarlet fever cases.   A. Knyvett Gordon[27] claims that the ear infection in scarlet fever may be through the general blood stream.   The postmortem investigations of Strauch[28] may be of interest in this connection.   He demonstrated streptococci in the blood in 79 per cent of cases of scarlet fever.   In measles the blood contained bacteria in 64 per cent.   In diphtheria, organisms were found in the blood in 45 per cent of the cases.   In meningitis from all causes 50 per cent had positive blood cultures.   In propagated meningitis, half the cases being due to ear infections, organisms were found in the blood in over 84 per cent, pneumococci and staphylococci being the predominating types.   An extension of the infection to the general circulation may also result from a secondary meningitis, a brain abscess, an extradural abscess, or even from a metastatic focus in a distant part of the body. It is essential to remember that these are exceptional instances, but they seem to demonstrate that a positive blood culture must not be accepted alone as positive evidence of a sinus thrombosis.

In the large majority of cases an infection of the general circulation following acute otitic disease means a thrombosis of the sigmoid sinus, but the bacteremia is accompanied by classic clinical symptoms as described by McKernon,[29] and the diagnosis rests on these symptoms in the presence of a bacteremia and not on the bacteremia alone.

(a) Positive cultures.—A positive blood culture following acute otitic infection indicates a general systemic infection of otitic origin, excepting the instances where the bacteremia is

due to a current infectious disease, scarlet fever for example, or possibly to a complicating infection in another part of the body, pneumonia for example. Finding the same organism in the blood that was previously demonstrated in the ear is corroborative but not necessarily positive proof of otitic general infection. As streptococci are often found in the blood in scarlet fever, a streptococcemia found with a complicating otitic infection in this disease cannot necessarily be ascribed to otitic origin of the general infection. Positive cultures with a clinical picture of pyemia usually indicate thrombosis of the sinus and form the large majority of the general infections of otitic origin in which metastatic foci are common. Positive cultures with a clinical picture of septicemia indicate a general infection without thrombosis of the sinus and are exceptional. Metastatic foci are rare. Some are quite mild infections and are overcome without difficulty. Others are most virulent and rapidly fatal with characteristic lesions in the heart, kidney, spleen and other organs.

(b) Negative cultures.—While negative blood cultures are uncommon when sinus thrombosis has occurred, they are possible, and for this reason repeated cultures are essential if suspicious clinical symptoms exist. McKernon[30] states that in the presence of typical clinical symptoms of sinus thrombosis it is not always wise to wait for a positive blood culture; at the same time the routine use of blood cultures would probably often avoid unnecessary sinus exploration.

A thrombus may be quite firm and may contain but few organisms, and even, if infected to a greater extent, its firm character may prevent infection of the general blood stream. It is also possible for detached portions of an infected clot to cause metastatic foci in distant parts of the body without infecting the general circulation. When suggestive symptoms occur, a negative blood culture may for the moment be used as an argument against sinus thrombosis, but if these symptoms continue, repeated cultures are necessary, as previously stated, and the former negative culture can have no weight whatever in the diagnosis.

4. Conclusions.—Acute otitic infections are due chiefly to the streptococcus, with the other more common organisms in the following order: Staphylococcus, pneumococcus, streptococcus mucosus, etc., A prognosis concerning the probability

of extension or virulence of the infection is not justified by
the type of infection.   A bacteremia denotes infection of the
general blood current, but in itself is not sufficient for a diag-
nosis of sinus, bulb or vein involvement.   While sinus throm-
bosis is the most frequent cause of positive blood cultures in
the cases under consideration, exceptions are too frequent to
justify such conclusion.   Cases of bacteremia with and with-
out sinus phlebitis present different clinical pictures, and the
positive blood culture does not aid in the differential diagno-
sis.   Negative blood cultures do not necessarily exclude a sinus
thrombosis, and suggestive symptoms should call for repeated
cultures.   Intercurrent diseases or complications of the orig-
inal causative disease with fever may occur during the course
of the otitic infection.   The blood cultures may aid in the dif-
ferential diagnosis as detailed, but careful clinical observation
and other laboratory aids as indicated are often of even greater
service.   Acute articular rheumatism shows a negative blood
culture, while a pyemic joint is usually associated with a bac-
teremia.   Pneumonia is usually accompanied by characteristic
physical signs, and possibly a bacteremia of other type than
the otitic infection.   Typhoid shows a leucopenia and relative
lymphocytosis, with possibly characteristic organisms on blood
culture and a positive Widal reaction; while an extension of
the otitic infection shows a leucocytosis and relative polynu-
cleosis, with possibly a positive blood culture of the organism
found in the ear.   Tuberculosis also shows a leucopenia and
relative lymphocytosis, and even a positive diazo reaction at
times, but the blood cultures are sterile and a von Pirquet may
be positive.   Fluctuating temperature, headache and local neu-
ralgia, due to an unrecognized acute nephritis, may develop
during the convalescence of a scarlet fever mastoid.   As a
streptococcemia is not infrequent in this disease, its discovery
might lead to a diagnosis of sinus phlebitis, if the rule is
accepted that a bacteremia invariably indicates this condition.
The value of the leucocyte count, particularly of the differential
count, in surgical otology, as previously detailed by McKer-
non[31] and myself,[32] has recently been employed by Urbant-
schitsch[33] in a careful analysis of seventy-five cases.   He also
attaches much importance to the determination of the coagula-
tion period of the blood, and details twenty-three cases.   In
all cases with sinus thrombosis the coagulability was increased,

whereas in those with general infections without sinus thrombosis, the coagulability was retarded.

REFERENCES.

1. Libman: Arch. of Otology, Vol. 37, No. 1, 1908.
2. Hasslauer: Internat. Centralbl. f. Ohrenheilk., Vol 5, 1907.
3. Urbantschitsch: Lehrbuch d. Ohrenheilk., fifth edition, 1910.
4. Dench: Annals of Otology, Vol. 12, 1903.
5. Alexander: Monatsch. f. Ohrenheilk., Vol. 39, p. 247.
6. Rolly: Münchener med. Woch., 1, 1911, 17.
7. Hoffman: Monatsch. f. Ohrenheilk., Vol. 39, p. 576.
8. Desguin: Memoir es coursonnes, etc., 19, 1908.
9. Meyer: Münchener med. Woch., 6, 1910, 300.
10. Carr: Archives Pediat., January, 1910.
11. Adams: Lancet I, 1910, 292.
12. Claude Sourdel: La Progres Medical, 17, 1910.
13. Leutert: Arch. f. Ohrenheilk., Vols. 46 and 47, 1899.
14. Hoffman: Zeitschr. f. Ohrenheilk., Vol. 50, 308.
15. Chmourlo: Arch. Internat. d. Laryngol., etc., Vol. 30, 1910, 1005.
16. S. Oppenheimer: Zeitschr. f. Ohrenheilk., Vol. 63, Heft 4, 1911.
17. H. Luc: Annales d. Mal de l'Oreille, Vol. 35, No. 6, 1909.
18. Rimini: Arch. Internat. d. Laryngol., etc,, Vol. 28, No. 4, 1909.
19. Gilbert: Annales d. Mal. de l'Oreille, Vol. 35, No. 12, 1909.
20. Zebrowski: Monatsch. f. Ohrenheilk., Vol. 40, 1906.
21. F. Müller: Monatsch. f. Ohrenheilk., Vol. 43, 1909.
22. R. v. Wild: Zeitschr. f. Ohrenheilk., Vol. 57, 1909.
23. Kobrak: Archiv. f. Ohrenheilk., Vol. 60, 1904.
24. Hayman: Archiv. f. Ohrenheilk., Vol. 83, Heft 1 and 2, 1910.
25. Duel and Wright: Transacts. of Amer. Otol. Society, Vol. 11, 1911.
26. Bennecke: Monograph, Jena, 1909.
27. A. Knyvett Gordon: Practitioner, New Series, Vol. 30, 1909.
28. Strauch: Zeitsch. f. Hyg. u. Infektsionskrankh., Vol. 65, 1910.
29. McKernon: Laryngoscope, July, 1905; New York Med. Journ., July 1 and 8, 1905.
30. McKernon: Yale Medical Journal, March, 1912.
31. McKernon: New York Medical Journal, January 19, 1907.
32. Sondern: Annals of Otology, September, 1910.
33. Urbantschitsch: Monatsch. f. Ohrenheilk., Vol. 45, 1911.

THE PATHOLOGY OF THE EUSTACHIAN TUBE—
A CLINICAL STUDY.*

E. M. HOLMES, M. D.,

BOSTON.

Were it not for the resulting aural complications, except for very rare malignant disease, the eustachian tube, physiologically or pathologically, would be of little importance, but the mucous membrane lining the tympanum and mastoid cells absorbs air, and as there can be little difference between the density of the air within the tympanum and that of the surrounding atmosphere without loss of function, a normal ear depends upon a patent eustachian tube, and becomes almost immediately affected when for any reason air cannot pass through the tube. It is in infectious and suppurative processes of the nose, of the epipharynx and of the eustachian tube that nearly all of the suppurative processes within the middle ear have their origin. Practically all of the serious complications of the mastoid, of the labyrinth and of the intracranial extension have their origin within the upper respiratory organs and the eustachian tube. Infection can take place in the middle ear through the eustachian tube and cause slight disturbance and slight symptoms if only the tube remains sufficiently clear to properly ventilate and drain the aural cavity; but often the tube becomes blocked and the excessive secretion is not drained from the tympanum; the accumulated secretion causes pressure, and immediately there is a hindrance to the circulation of the blood to and from the inflamed area. This condition reduces to a marked degree the natural resistance as well as the processes of repair. We have demonstrated this by our studies upon acute suppurative processes within the middle ear, treated by relieving the inflammation within the eustachian tube. It is evident that nearly all of the se-

---

rious middle ear complications are due to previous and coincident disease within the eustachian tube, and it is also generally conceded that over 90 per cent of all aural disease is due to trouble in and about this tube. Since the beginning of otology it has been the aim of the aural surgeon to relieve the conditions in the middle ear caused by the partial vacuum, by forcing air through the tube. Much relief has been given by the Valsalva and Politzer methods, and also by the catheter, but in many cases relief has been but temporary, for the underlying causes of the tubal stenosis still remained. The study of rhinology and the treatment of nasal diseases has done more to relieve conditions in the epipharynx and eustachian tubes and to restore the function of the ear than any other effort, but although relief from pathologic conditions in the nose can cure many cases of eustachian tube disease, there are many which are not relieved, and which continue to grow worse after the nose has been as fully restored to its normal condition as our present knowledge permits. There are also a large number of cases of tubal disease where we can find no pathologic conditions within the nose. Many of these cases, without direct treatment of the tube, progress and produce the marked chronic inflammations within the middle ear, resulting in deafness, often accompanied by distressing tinnitus, and sometimes by vertigo.

Until the small mirror was introduced for reflecting light into and obtaining a reflected image of the epipharynx, little was done toward studying this area in the living. There are many drawbacks to the successful use of the mirror, the greatest obstacle being the soft palate. If this curtain is carried forward by any of the many hooks, the eustachian tube as well as the surrounding tissue is more or less distorted, and it is impossible to observe the normal movement in and about the tube during deglutition. Under the most favorable conditions the oropharynx is not a satisfactory viewpoint. This is true in regard to any instrument passed through the mouth. In order to study the epipharynx and eustachian tube, we must illuminate this area and by the aid of prisms and lenses carry our point of view within the epipharyngeal cavity. The nasopharyngoscope performs this duty, and when it is passed through the nose the whole epipharynx and pharyngeal end of the eustachian tube can be thoroughly examined. There is no gagging

or annoyance from the soft palate, and the movements of the structures in and about the tube during deglutition are easily studied. Growths, bands and adhesions in the lateral fossæ are often not noticeable until the tube is pulled forward and downward by the palatal muscles. There are also restrictions of movement of and within the tube which cannot be diagnosed in any other way.

Every movement of instruments used to operate upon or treat the tube and passed through one side of the nose can be followed and directed by vision obtained by the aid of the nasopharyngoscope passed through the opposite nostril.

In November, 1910, I modified the small cystoscope, and later designed an instrument which gave a larger field and is much smaller and lighter than any instrument designed up to that time. With this instrument I began my studies of the eustachian tube. In February, 1911, I demonstrated the instrument by showing a series of pathologic conditions of the epipharynx and eustachian tube at the meeting of the Eastern Section of the American Laryngological, Rhinological and Otological Society.

I have now examined nearly two thousand cases, the majority of whom sought relief from some aural disease. Although I have made considerable advance in my technic for examination and in the general knowledge of this study, there are so many variations and so many degrees in pathologic conditions, and so many and varying degrees in the associated aural pathology, that I feel we have merely made a beginning. Concerning the more marked pathologic changes and their sequelæ, we can speak with a greater degree of authority, and it is my object in this paper to present in a general way what we have been able to demonstrate.

There are cases which show faulty ventilation of the tympanum, where the tube upon first inspection shows slight if any changes. Often, however, if the cases are examined several times, there will be found at some time a marked congestion of the membrane, or some restriction of movement which was not present at the previous examinations; or perhaps there are at times hypersecretion of mucus sufficient to hinder normal inflation while it lasts, and at other times the mucous membranes and tubes may appear normal. The epipharynx and tubes also vary to such a degree without producing symptoms, it is not

always easy at first to say positively if we have a physiologic
or a pathologic variance. The one important question is, does
the existing condition interfere with the ventilation of the
tympanum, or is it liable to produce infection of that cavity?

Viewed by the aid of the nasopharyngoscope the epipharyn-
geal end of the eustachian tube appears as a more or less
rounded triangular-shaped opening, bounded behind and above
by a rounded curved eminence. This eminence is called the
cushion of the tube, and is the pharyngeal end of the tubal
cartilage. Extending anteriorly from the cartilage, and form-
ing the anterior wall of the tube, is a fold which extends down-
ward into the soft palate. In front of this fold is an area
which is directly over the sphenopalatine fossa, and is of
interest, as through this membrane emerges a plexus of vessels
which are liable to become markedly engorged after any irrita-
tion to the epipharynx and tube. In chronic inflammatory
processes these vessels are usually much dilated. In front of
the sphenopalatine area are the posterior ends of the turbinates.
The turbinates are frequently found so hypertrophied as to
cover and press upon the anterior lip of the eustachian tube.
During the act of deglutition the anterior lip of the tube be-
comes tense, the cartilaginous posterior superior portion of the
tube is rotated, the cushion of tissue at the floor becomes tense
and the opening of the tube appears for the moment more
rounded. These motions are almost immediately reversed and
the structures resume their original relation.

Although there is always a general similarity of the move-
ments in and about the tube during deglutition, there are
marked differences in degree apparently within limits of nor-
mal function. A large patent tube will naturally give better
ventilation with less movement of the walls than a narrow
constricted one. An occlusion or partial stricture within the
tube may prevent the function of inflation, while the epipharyn-
geal opening as well as the structures in and about this opening
are normal in structure and movement. A redundance of
mucous membrane or a plug of thick tenacious mucus may
often block and absolutely prevent the function of a tube which
is otherwise anatomically normal.

In posterior purulent ethmoiditis and in suppurative sphenoid
disease the purulent secretion runs downward over the poste-
rior end of the middle turbinate and over the opening of the

tube, and pus within the tube can often be traced to its source in these sinuses. Cardiac, renal, gastric and intestinal diseases often produce marked changes of the mucous membranes within the tube. These membranes also often become congested in vasomotor disturbances, as at the menses and the menopause.

Intemperance in smoking and drinking alcoholics play no small part in the condition of the epipharynx and tube. Rheumatism and gout are also frequently etiologic factors. We have seen three cases of spongy, somewhat edematous looking tubes where there were signs of lead poisoning, and all of these cases were relieved by a course of treatment for this condition. A diabetes with even a small amount of sugar can produce a marked inflammatory process in the fossa and tube. We have seen seven such cases. In all cases it is of the utmost importance to know first of all the general condition of the patient, and if any organic disease is producing or influencing the local condition, that, if possible, must first be relieved.

The same inflammatory processes are found in the eustachian tube as in other mucous membranes, and they vary from slight hyperemia to intense inflammatory swelling, from marked hypertrophy to pronounced atrophy, and from a parched dryness to excessive mucopurulent secretion, at times forming crusts.

Acute inflammation of the tube is a very frequent occurrence. In the large majority of all infections of the nose there is an extension into the epipharynx and also into the tube. The early symptoms of this extension are not necessarily severe in an otherwise normal tube, and only when the swelling or secretion has become sufficiently marked to obstruct the opening and prevent the passage of air do the symptoms become at all severe and distressing. The first noticeable change in a beginning inflammation is an injection of the mucous membrane similar to that seen upon the nasal septum at the beginning of an acute rhinitis. This becomes more intense, there begins to be a noticeable swelling and after a few hours the whole membrane becomes puffed and irregularly swollen. The blood vessels emerging from over the sphenopalatine area become engorged and prominent. The motions of the tube become restricted and the swollen lining membrane is in contact during deglutition and mechanically prevents the ventilation of the ear. This acute inflammation of the tube, if not accompanied with marked

swelling or if in a tube of large lumen, may subside without having produced aural signs or symptoms, and in mild attacks there is a return to a normal appearance by the end of a few days.

A more severe type of inflammation may begin and appear at first as the simple acute. The swelling usually becomes more marked and there is usually a mucous or mucopurulent secretion. The secretion at first is of a mucous character, but after a few hours it is found to be of a purulent character. It can often be seen as a plug in the lumen of the tube, or extending from its orifice. The lateral fossa is usually attacked early in the disease and is frequently found injected and swollen before the tube shows any abnormal signs. On account of the glandular structures in this fossa we more frequently find excessive secretion and swelling, which often limits the motion of the tube to a marked degree, thus preventing its function, before any marked inflammatory process has taken place within its lumen. Upon first inspection the only noticeable change may be that of a narrow lateral fossa. As soon as the patient swallows it will be noticed that there is a restricted movement of the cartilaginous portion of the tube, and secretion will be forced from the lateral fossa. The symptoms of this condition are often those of a snapping tinnitus, more marked during deglutition. There is frequently a sense of itching within the ear and sometimes extending into the throat. Soon there is a sense of fullness and pressure associated with more or less pain. These symptoms are due to the middle ear complications and can almost always be relieved by relieving the congestion within the tube. Acute purulent salpingitis is usually accompanied by more marked stenosis of the tube, and more frequent middle ear extension which is usually of a purulent type. With a closed and septic tube, where drainage as well as inflation is suspended, it is surprising that any middle ear escapes being suppurative. In this condition the mucous membrane covering the tube becomes much swollen, the glandular structures are frequently more swollen than the surrounding tissue and appear as elevated nodules. The whole mucous membrane has a granular appearance. These granules are more prominent within the orifice of the tube. More or less pus is found upon the mucous membrane, and after relieving the swelling of this membrane within the tube by applying cocain and

adrenalin, there is frequently found thick, tenacious, purulent secretion completely filling the lumen.

Acute purulent salpingitis is of great importance, for this is the frequent cause of purulent middle ear disease, with all the complications which may accompany and follow that condition. Acute purulent salpingitis, when recognized early, is amenable to local treatment, and even when there has developed an inflamed middle ear, with secretion and the resulting processes, causing a bulging membrane and pain, the tube can in the great majority of cases be sufficiently restored to function to give drainage to the middle ear secretion and relief to the distressing symptoms. By early recognition of and careful attention to the treatment of the acute purulent eustachian tube, many of the resulting purulent inflammations of the middle ear can be prevented and relieved. This means that many of the suppurative ears which have in the past produced serious destruction of important structures, with resulting loss of function, and in many cases have demanded serious operation to save life, can be prevented.

Frequent acute inflammations or long-continued inflammations may produce many pathologic changes; the more frequent condition is that of a general hypertrophy. The cushion of the tube is not only swollen and spongy in appearance, but frequently the underlying cartilage becomes enlarged, probably a true chondritis, the cushion is frequently in contact with the posterior border of the lateral fossa, and often there are adhesions between it and the lateral pharyngeal wall. The anterior lip of the tube, which is the fold of mucous membrane covering the tensor palati muscle, becomes thickened, and except for the color, which is usually of a reddish tint, appears similar to an edema of the uvula, or that occurring in the mucous membrane within the larynx. The mucous membrane at the floor of the tube has much the appearance of that which covers the anterior lip. It is swollen and forms a much more prominent crest than is normal. This is more noticeable during the act of deglutition. In nearly all of the chronic hypertrophic inflammatory conditions of the eustachian tube there is an excessive secretion, which varies much in character. More frequently it is of a glairy, tenacious mucus, and is found to form air bubbles within the orifice of the tube, and especially within the lateral fossa. These bubbles within the tube are

seen to absolutely prevent the passage of air when otherwise
the tube is not impervious. In almost all chronic in-
flammatory processes of the tube the blood vessels which
emerge from the sphenopalatine area and run backward over
the anterior lip of the tube become enlarged. They remain
large after the application of cocain and do not contract as in
acute inflammations. The glands underlying the mucous mem-
brane, both upon and within the tube, may become greatly
hypertrophied, and when this has taken place the surface has
much the appearance of a miliary tuberculosis of the splenic or
kidney capsule.

Less frequently we find a chronic inflammatory process pre-
senting a very different appearance. Entirely different changes
have taken place, as instead of a hyperplasia we have an atrophy
of the mucous membrane of the glandular underlying tissue,
and at times apparently more or less atrophy of the cartilagi-
nous wall. There is a general glazed appearance of the mucous
membrane, and there are frequently crusts of secretion in the
lateral fossa, and at times within the lumen of the tube. The
atrophic salpingitis is the most serious form with which we
have to deal. Frequently there is an associated purulent mid-
dle ear of a chronic type. Treatment in the severe types of
this disease has been of little avail. In the majority of the
cases of atrophic salpingitis there is also an associated or pre-
existing atrophy of the nasal mucous membrane. There are,
however, a few cases where the nose is apparently normal.
We have seen two cases of unilateral atrophy associated with
chronic purulent middle ear of the same side, which may have
been due to the constant flow of pus from the tympanum
through the tube. There is occasionally found a condition
resembling somewhat the atrophic salpingitis, where pus from
the posterior ethmoid or sphenoid discharges over the tube and
forms crusts very similar to those found in the true atrophy.
In these cases, after cleansing the area, the mucous membrane
is found to have more the appearance of chronic hypertrophy.
This class of cases yields readily to treatment as soon as the
source of irritation has been removed.

Both in adults and in children adenoid hypertrophy about the
tube and even within the lumen is frequently found, but neo-
plasm is comparatively rare. I have found but two cases in a
series of about two thousand examinations. One growth which

completely filled the lumen of the tube was about the size of an orange seed. This was pathologically a myxofibroma, and judging by the aural symptoms the growth was at least of five years' standing. Another growth which was much larger than the first was also attached within the lumen of the tube, and protruded from the orifice some quarter of an inch. This growth was found to be simple fibroma.

I have found four syphilitic ulcerations in the tube, and one syphilitic swelling of the cartilaginous cushion. The ulcerations appeared similar to the same condition upon the pharyngeal and buccal mucous membrane. Three of these cases gave a history and other signs of syphilis, and the other gave a positive Wassermann and yielded to salvarsan.

We have seen one case of lupus which covered the whole velum palati and extended onto the floor and anterior lip of the eustachian tube. Although I have examined quite a large number of patients who were suffering with tuberculosis, this is the only case where there was found any signs of tuberculosis about the eustachian tube.

The most common cause for impairment of function of the eustachian tube is undoubtedly adenoid hypertrophy. It is surprising to find how frequently small amounts of adenoid tissue in the lateral fossa and upon the cushion of the tube cause a chronic congestion of the whole tube. It has impressed us to find how frequently these small collections of adenoid are found about the tube after operations performed by careful and skillful surgeons. This explains the comparatively large number of patients who are not permanently relieved of the ear symptoms by the adenoid removal. The only way to thoroughly relieve these cases is to remove the small masses with a sharp curette or a small cutting forceps, under the guidance of vision obtained by the nasopharyngoscope.

Next to adenoid vegetation in frequency we have found hypertrophy of the posterior end of the turbinate to cause restricted movement of the tube, as well as irritation due to contact with the anterior portion of the tube. In some of these cases the mechanical effect of the turbinal hypertrophy is not apparent until the patient swallows. In many of these cases the simple removal of a small portion of the posterior end of the turbinate has resulted in as marked improvement of the tube and the aural complications as when excessive adenoid tissue

has been removed from about the tube. It is a simple thing, now that we can see the protruding end of the turbinate through the nasopharyngoscope passed through the opposite side of the nose, to remove as much—and only as much—of the hypertrophy as we wish with a snare passed through the same side as the existing hypertrophy.

These pathologic conditions have been known for many years, and in a general way have been described by many otologists. Not until we were able to see the effects of these abnormal conditions through an instrument which does not of itself distort or restrict the physiologic functions, were we able to determine clinically the extent and result of many of these pathologic conditions; and being able to treat these conditions under guidance of vision, we are enabled now to relieve many of them which we otherwise could not; and as our knowledge increases and our technic improves, we shall in all probability relieve many more than we can at present.

LXIII.

THE PATHOLOGIC AND CLINICAL SIGNIFICANCE OF SOCALLED AURAL AND NASAL POLYPI, AND THE ETIOLOGY OF BENIGN TUMORS IN GENERAL.

By Prof. Citelli,[*]

Catania, Italy.

By polypi of the ear is understood new tissue formations which tend ordinarily to increase in volume slowly but continually. In the majority of cases they have their origin from the tympanic cavity and sometimes from the walls of the auditory canal. They are of a reddish color, occasionally grayish white, and are accompanied by a seropurulent or purulent discharge.

Does this new tissue formation, which presents a different color, appearance and histologic structure, always have the same pathologic significance, or is it advisable to differentiate several varieties of aural polypi? This appears to me to be a question of great importance from a pathologic and clinical standpoint, and one which I shall try to solve on the basis of the pathologic and clinical study of a large number of cases.

The socalled polypi are commonly considered as granulomata or new formations of granulation tissue, caused, as a rule, by chronic suppuration of the tympanic cavity. Some authors (Brühl, Politzer, etc.) divide aural polypi into two classes: first, polypi formed by granulations (called by Brühl, polypoid granulations, and by Politzer round-celled polypi), and second, polypi which represent true benign tumors (fibroma, fibromyxoma, etc.). According to Brühl, there is no etiologic relationship between the one class and the other. By polypi I mean the common form, and not tuberculomata or malignant tumors, which may occasionally take on the macroscopic appearance of fleshy polypoid growths. It is clear, pathological-

ly and clinically speaking, that a fibroma which begins as such is a totally different thing than a conglomeration of granulation tissue. In fact, polypoid granulations are pathologically granulations and are clinically the result of a lesion of the walls of the tympanic cavity and of the auditory canal, and are often of a serious nature; therefore, the prognosis should be guarded. Fibromata and fibromyxomata of the ear, according to Brühl, may often have their origin in an otherwise healthy ear, and their removal is usually followed by a complete cure.

Having studied numerous cases of aural polypi, I will briefly mention eight such cases, which are very important for our statistics, and offer certain facts regarding their structure which may help to solve the question of the pathogenesis and etiology of benign tumors in general.

Case 1.—Polyp of the superior wall of the auditory canal.

Clinical History.—Young woman, healthy; family history negative. Slight serous discharge in left ear, no pain. The relatives discovered a mass protruding from the auditory canal.

Examination.—I found a protruding polyp of grayish color surrounded by scanty serous discharge. Upon removal with cold snare I found that it sprang from the superior wall of the auditory canal; bone rough. The polyp was of a cylindrical shape, 12 mm. long, with a broad flattened base and a short pedicle.

Structure.—The extreme external portion of the polyp was formed principally by delicate connective tissue, fixed cells, and few vessels; and occasionally areas of granulation. Pedicle formed by granulomatous tissue. The whole was covered by stratified epithelium. It is apparent that the external portion was fibromatous in structure, the pedicle granulomatous.

Case 2.—Woman with a polyp of the tympanic cavity which extended to the external meatus. It has a narrow pedicle and is attached to the posteroinferior quadrant of the labyrinth wall. Scanty seropurulent discharge for a number of years. Removed with cold snare, curetted the pedicle and cauterized with trichloracetic acid. Healing in three weeks. Hearing diminished (whisper 1 m.). The polyp was of a cylindrical form, whitish color, 2 cm. long.

Structure.—As in case 1.

Case 3.—Young man, age twenty, with purulent otitis media

since the age of four. Spontaneous detachment of the polyp of the size and shape of a bean.

Structure.—Epithelium stratified with horny degenerations. No papillæ, but considerable areolar tissue with fixed cells. Round cell infiltration around small and numerous vessels. This was clearly a soft fibroma.

Case 4.—Young lady, age fourteen, with polyp and puriform secretion for four years. The polyp, which reached the external meatus, was of a cylindrical form 2 cm. long, attached to the promontory. It was smooth, grayish white in color, with a granular pedicle. I removed it in the usual way.

Structure.—Same as in case 1, with myxomatous degeneration of the external portion. Numerous vessels, no round cell infiltration. The pedicle granulomatous, as usual.

Case 5.—Child, age eleven, with adenoids and purulent otitis media for a number of years, with a polyp of the ear and nasal polypi. Adenoids and polyp were removed. The latter was of a grayish color, cylindrical in shape, 2 cm. long. This polyp was attached to the promontory and healing was not complete a year after operation. This polyp is mostly of myxomatous tissue, surrounded by a thin dense fibrous capsule, with few vessels and cells. The pedicle, as usual, granulomatous. This is a case of pure myxoma, which has certainly prolonged the suppuration of the ear.

Case 6.—Obese woman, with a large polyp protruding from the meatus. This was removed and found to have been attached to the promontory. It was 2½ cm. long and boggy, as if it were a cyst. Color grayish white; healing complete in a month. This case is similar to case 3, but with large cavernous sinuses scattered throughout. The polyp was a fibromyxoma or fibromyxoangioma.

Case 7.—Woman, age thirty, healthy, with prolonged tympanic suppuration of the right ear. Tympanic membrane almost entirely destroyed, labyrinth wall cicatrized, and springing from the oval window was a small tumor of the size of a large bean. This I removed. The color was reddish, surface smooth and surrounded by a slight serous discharge proper to the tumor. Complete healing followed a few days after this small operation.

Structure.—This tumor resembled cases 3 and 6 histological-

ly, and it should be considered as a soft fibroma due to the prolonged suppuration of the ear.

Case 8.—Man, age thirty, suppuration from the tympanic cavity for a number of years. A small round polyp of the size of an ordinary bean, with smooth surface, was attached behind and beneath the promontory. Scanty serous secretion. Removal. Length 14½ cm. It presented areas of myxomatous degeneration.

REMARKS.

The above eight cases must be considered as fibromyxomata and myxomata, but not as granulomata, inasmuch as the fundamental tissue is mostly areolar connective or myxomatous, similar to that found in nasal polypi. Before concluding whether the above eight cases of polypi of the ear should be considered as tumors, I wish to insist on a fact worthy of the greatest consideration, that is, the distribution of the granulomatous tissue. This seems to have formed the greater portion of the root of the tumor, which gradually diminished or rapidly disappeared toward the extremity or base. (As in cases 7 and 8.) In some of the cases without any continuity of tissue there was found in a limited area of the periphery of the fibroma or fibromyxoma, a certain quantity of granulomatous tissue, readily distinguished by the naked eye on account of its red color and smooth surface. (Cases 5, 7 and 8.) In case 4, where the tumor extended to the meatus, this change was marked, but the granulation tissue continued into the fibromatous tissue. In other cases (1 and 4) the disposal of the vessels and other characteristics of structure, plainly showed that there must have been previously granulomatous tissue in the fibroma and fibromyxoma. Now then, the constant presence of granulomatous tissue in the above tumors, and particularly the distribution of the same, has a great bearing on the pathologic significance, as well as on the important question of the pathogenesis and the etiology of benign tumors in general. The attachment of these polypi to the mucous membrane of the tympanum or of the auditory canal was frequently formed by granulomatous tissue, and in addition it was always possible to discover denuded bone. The tympanic suppuration was always of long standing and often persisted long after the removal of the tumor. From these facts the following conclu-

sions may be drawn: The point where the tumor is inserted probably began with the pathologic lesion of the wall by means of granulation tissue. As seen in the pedicle, the proliferation of the cells is more recent and active (because in contact with the most diseased part) and keeps its original character.

Further away from the point of insertion, on account of the disposition of the individual and the nature of the infection, which acts as an irritating cause, and the edema of the tissue from stasis due to the compression of the pedicle, the free end of the small granuloma begins to undergo a fibromatous transformation. The granulation tissue becomes organized as in scar tissue, but forms connective tissue instead. The extreme free end of the tumor as it gets further away from the point of origin being of a later formation and less irritated by pus than the internal portion, has time to become transformed into connective tissue, even at the periphery. It will then be found, as in many of our cases, that the whole or almost the whole portion of the blood vessels shows the previous existence of granulations, the cells of which are partly destroyed or transformed. But there is another important consideration based on the presence of small isolated zones of granulation tissue at the periphery of the tumor (cases 4, 7 and 8). These areas are frequently papillomatous and partly uncovered by epithelium, whereas, the rest of the tumor is fibromatous, which according to me is due to external irritations, i. e., manipulation, collection of pus and other causes. These, therefore, must have been formed by fibromatous tissue, and subjected to these intense irritations. This fact, together with the transformation of the granular tissue into the fibromatous, shows the intimate relationship between the two; it is therefore quite possible under favorable conditions that the one may readily be transformed into the other.

After what has been said, should the above polypi be considered as tumors in the true sense, there being in many of them the remains of granulation tissue? The pathologist distinctly defines the new tissue formation of inflammatory origin (as granulations) and those such as tumors (benign and malignant). It has been demonstrated by facts, however, that the etiology of tumors, of which little is known, is influenced by irritants and inflammations in general. As the same irritating causes may produce inflammatory changes as well as tumors,

and as it has been seen that inflammatory tissue may pass into a neoplastic formation, it is quite plausible to have new tissue formation which may present the characters of inflammation and those of a neoplasm; and it also explains why a doubt may arise from a microscopic examination, as to whether new tissue formations are inflammatory products or tumors. The cases described by me are of the mixed type, having the characters of both inflammation and tumor. But as the predominating type of structure in these cases was of a neoplastic order, and as they continually increased in size, we cannot but consider them true tumors. Although these tumors have a secretion proper to themselves, they are as a rule secondary to a suppurative inflammation. This is also shown by the clinical history and the structure of the tumors, as well as the evident results of chronic suppuration of the tympanic cavity which is often found. After what has been said, the sprouting of the above mentioned tumors can be explained by admitting a limited area of inflammation of the walls of the canal. This beginning of the inflammatory type would change by chemical process and a predisposition into a neoplastic type; if the special conditions, on the other hand, which determine the predisposition are not present, the granulations would never become transformed into tumors. To uphold the theory of inflammatory pathogenesis of these tumors of the ear, there is also the fact that for all tumors in general may be added the influence of irritants which in the beginning will probably give rise to inflammatory conditions and from which the tumor may originate. Therefore, the great importance of the etiology of benign tumors is at present neglected, as every effort has been directed to the solution of the practical one of malignant tumors.

From the structure and clinical characters, two kinds of aural polypi should be considered: first, polypi of the type of granulomata; second, polypi of the neoplastic type (tumors). The first are of a reddish color, granular surface and are composed mostly of granulation tissue. The second kind is of a grayish white color, smooth surface and have the structure of fibroma, fibromyxoma and fibroangioma. I have noticed, however, that granulomata which remain as such are frequently found with intense and diffuse lesions of the tympanic cavity, whereas in the polypi of a neoplastic type the prognosis is

favorable because the tympanic lesion is slight and circumscribed. This appears to be the reason why the inflammatory elements, not being subjected to strong stimuli, are readily transformed into fibrous tissue. Besides the intensity of the lesion of the tympanic cavity, age has a decided influence on the type of polypi. In children, therefore, the connective tissue reacts readily to stimuli and almost always granulations are found. I will add, to uphold the theory of the inflammatory pathogenesis of neoplastic aural polypi, that in several patients the benign tumor of the canal started from the site of a furuncle. It is interesting to note that from a pathologic as well as a clinical standpoint there are many analogies between the common aural and nasal polypi. In the nasal fossæ, in fact, besides the formation of polypi following purulent sinusitis and ethmoiditis, these polypi which are apparently primary are in reality secondary to catarrhal inflammation of the ethmoidal cells. Even those cases where the inflammation was not very evident, it probably existed and was of a slight and fleeting nature. Even in the common nasal polypi the distinction between the inflammation and neoplastic forms is not always distinct and there is an uncertainty at times as to whether the polyp is the result of inflammation or a benign neoplasm; because the intermediary stage is gradual and the one goes into the other. Then again, as in soft fibromata, there is at the root or some part of the periphery subject to special irritation, a granulomatous rather than a fibromatous structure. The above considerations will sufficiently uphold my views regarding the etiology and the histogenesis of benign tumors, i. e., that they originate from other tissues under various stimuli which are not specific, which according to Fichera has the greatest number of adherents, especially regarding malignant tumors. It is evident that this theory which admits of a common etiology and histogenesis for both benign and malignant tumors is one that satisfies us mostly, inasmuch as the distinction made between benign and malignant tumors is merely one of a practical character.

<div align="center">CONCLUSIONS.</div>

First.—That the common polypi of the ear may be divided into two classes: granulomata and neoplastic polypi—fibromata, myxomata, mixed forms. The first are of a reddish color, granular surface, composed entirely of granulation tissue, and

indicate a diffuse and severe lesion of the walls of the auditory canal. The neoplastic polypi are of a grayish transparency, have the structure of true fibromata, fibroangiomata, myxomata, and the prognosis is favorable. They are less frequent than the first class.

Second.—The first class as well as the second, contrary to Brühl, are always secondary to a latent or manifest inflammation of the walls from which they start, and can be transformed from one into the other class. If the structure of the various segments of the tumor is examined, one observes in some cases various phases of the transformation of the new growth from an inflammatory type, granuloma, to a neoplastic type. It is for this reason that one is occasionally in doubt as to whether or not the aural polyp should be considered as an inflammatory or neoplastic by-product.

Third.—Both aural and nasal polypi are well adapted for the study of the pathogenesis of benign tumors, and they make one think of a common pathogenesis caused by the influence of various inflammatory agents in tissues and individuals so predisposed.

Fourth.—The inflammatory histopathogenesis of benign tumors, which although in accord with the practical observations of centuries, i. e., regarding the influence of irritants as the cause of tumors, corroborates the histopathogenesis admitted also in malignant tumors. This proves satisfactory to us, inasmuch as it admits of a common pathogenesis for all tumors.

Fifth.—Contrary to Politzer's views, the epithelial covering of these aural tumors has scarcely any value as regards their point of origin, i. e., whether the tumor is attached to the canal or tympanic cavity; inasmuch as the eight cases studied by me had their origin from the tympanic cavity, and the epithelium covering them was of the stratified and not of the cylindrical variety.

LXIV.

TINNITUS AND DEAFNESS—A NEW INSTRUMENT FOR THEIR RELIEF.*

By TALBOT R. CHAMBERS, M. D.,

JERSEY CITY.

The photograph explains itself.

When in operation, a musical note is sounded synchronously with the vibration of the Victor pneumomassage pump. The note is not unlike the peep of a young chicken or like the chirp of a cricket. About three pounds of air pressure is employed; any greater pressure would prevent visible vibration of the drum of the ear.

In the case of a very depraved acoustic nerve, the patient turns the screw cap of the Galton whistle until that certain pitch is reached where the whistle is heard by the patient. The massage is continued with that note during the seance. The speed of the vibrations is kept at whatever rate is most agreeable to the patient. It is generally not very fast.

A seance consists of thirty seconds of suction-with-release with the whistle, followed by fifteen seconds of pressure-with-release, but without the whistle, in order to avoid exhausting the nerve. These two proceedings are repeated until five minutes have elapsed. The operator with finger on the stop cock from the air tank controls the whistle, while an assistant at the massage pump controls the lever for pressure or suction at command of the operator. The Siegle otoscope accurately fitted into the auditory canal allows the observer to see the drum during the whole seance. Before beginning the treatment the middle ear is inflated. An oil vapor is used, and in some cases the pharyngeal orifice of the eustachian tube is painted over with a weak idoglycerin. During the seance the patient is directed to forcibly close the eyelids and at the same time make the effort at swallowing.[1]

The theory of the treatment rests on the idea that in order

to stimulate a part of the body, its function must be exercised. A muscle to be stimulated must be contracted and relaxed. Bone which has been broken and fails to unite is forced to perform its function of resistance by the exercise of resistance. A stiff joint whose function is motion is relieved by passive motion. And so a nerve whose function is to recognize sound and has lost that ability must be exercised with such sounds as it will recognize, and later it will notice other and more complex sounds. A regiment of soldiers crossing a bridge is compelled to break step lest the vibration might break down or damage the bridge. The mastoid bone has similarly a note of vibration which the pneumomassage awakens, together with the parts included in the bone, which are blood and lymph vessels, nerves, endolymph and especially the terminal filaments of the acoustic nerve. Thus the vibration and rhythmic sounding of the note which the ear hears is a warrantable procedure for the relief of tinnitus and deafness, and though failures have been noted, there have been quite a number benefited sufficient to justify the claim that it is a valuable means of relief. According to Urbantschitsch and Marguilies, all hope need not be given up even in the very deaf.

The electromotor apparatus is not new. Dr. W. S. Bryant in 1905 proposed his phonograph acumeter.

Inflammatory conditions are a contraindication. Treatment should be repeated when the patient notices a subsidence of the good effects of a treatment.

Every case on which this paper is based had a tympanic drum. Many had had perforation.

Results of treatment by this method show:

1. Marked otosclerosis is but little benefited, and that but temporarily.

2. There may be no or little improvement to hearing the watch; yet the voice and other sounds may be greatly improved.

3. In a number of cases the improvement is marked and permanent, especially where the handle of the malleus is seen to move freely. Improvement has occurred in most unexpected cases, and failure has resulted where success seemed assured. Tinnitus may be relieved, and treatment show no improvement in hearing, and vice versa.

REFERENCES.

1. Fowler, E. P.: Manhattan Eye, Ear and Throat Hospital Reports, No. 11.

From Pneumo-massage Pump

Valve preventing air
returning from Drum

Finger Stopcock from
Compressed air tank

Galton whistle with
opening in drum
Screw cap raising or
lowering pitch
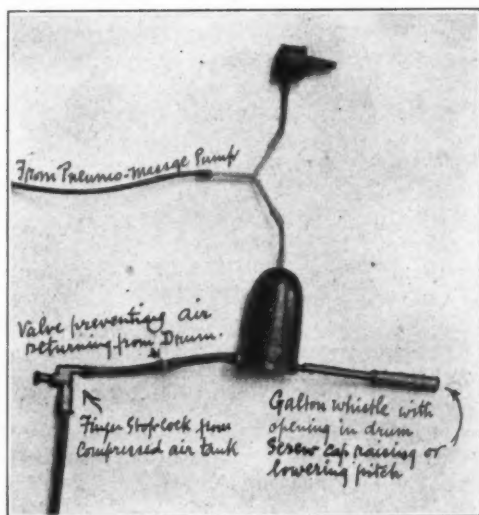
TREATMENT OF NASAL SYNECHIÆ WITH MICA PLATES.

By Prof. E. J. Moure,*

Bordeaux.

For a number of years rhinologists have been interested in methods of avoiding secondary adhesions, which occur occasionally after operations, between the turbinates and the septum, and destroying these synechiæ when their production could not be prevented.

To obtain this result the interposition between the raw surfaces of a tampon of cotton, iodoform gauze or something similar, has been successively advised. Then for the tampon was substituted flat plates of cardboard (Moritz Schmidt). The latter had the inconvenience of becoming easily impregnated with septic fluids and of getting just as fetid as the cotton, then softening and irritating the mucosa. Ivory plates have been utilized. In 1894, M. Molinie, in a paper before the French Society of Oto-Rhino-Laryngology,[1] took up the idea of Garel, who had recommended the employment of celluloid leaves, 1½ centimeters wide and 7 or 8 centimeters long. The straight upper edge, writes M. Molinie, should come forward anteriorly so as to lodge within the lobule of the nose. Rounding the angles facilitated the introduction of the plate without scratching the mucosa.

Garel had thought it advisable to perforate the anterior end, and fasten it with a thread to the ear during sleep. M. Molinie dispensed with the thread as useless, thinking the nasal secretions sufficient to hold the celluloid and prevent its falling into the nasopharynx.

The author added, as a concession to pride in some patients, that the plates could be rose colored to match the mucosa.

Since that time my Marseilles colleague has advised, in

---

*Translated by A. Miller, M. D., St. Louis.

order to broaden the instrument, to fasten two plates side by side with a wire in such a way that when introduced into the nose they could be spread apart like opening a razor, and held in place by another wire. This method, apparently so ingenious, did not give the results expected. It irritated the mucosa and often had to be abandoned. I will explain why further on.

In 1895, Wright advised the employment of gutta percha which could be cut with scissors to the desired shape.

Scheppegrell[2] went back to Garel's plan and also recommended celluloid plates.

Last year I heard a paper by M. Brindel, following the lead of M. Lavrand, of Lille, in extolling cauterization of the raw surfaces with chromic acid. This method really gives satisfactory results sometimes, but it cannot always be relied upon, for I have seen the plan fail in some patients.

To my mind the following are the reasons for the production of nasal synechiæ and the inefficiency of the methods employed for their prevention:

When we operate upon the turbinates, either with the galvanocautery or by resecting the mucous membrane, we know that the postoperative reaction is variable. In some patients there is formed upon the bared or burned surface a pseudomembranous eschar, which fills the whole cavity and forms a sort of adherent tampon, preventing union of the vivified surfaces, provided, of course, that the operator does not too soon try to remove the membranous pellicle. In these cases repair takes place under the pseudodiphtheroid exudate, and when the membrane falls off the two surfaces remain apart from each other, having no tendency to unite.

In other patients, on the contrary, the same surgical interventions are followed by a considerable swelling of the mucosa which inflames and suppurates and even has a tendency to granulate. In this class of cases the inflamed surfaces remain in permanent contact, and, in spite of assiduous watching and careful dressing, cicatrization is followed by adhesion of the opposing areas, when both sides have been by mistake or inadvertently freshened or irritated. Then come on the rebellious synechiæ, against which the various above mentioned procedures fail.

All interposed foreign bodies produce an irritating pressure,

preventing cicatrization and inflaming the mucosa. When the substance is removed cicatricial synechiæ are produced, or reproduced.

In these grave and rebellious cases even chromic acid does not give the results which it gives in other cases.

It was desirable, then, to find a substance which could be interposed between the surfaces without irritating them or preventing cicatrization, even in the unfavorable cases to which I have alluded. I believe that scales of mica give us the end which we seek.

These scales can be made extremely thin, thin as tissue paper. They are easily sterilizable, as mica bears very high temperatures without softening or breaking. Besides, being flexible, the plate may be introduced curled up through the nasal speculum. Thus introduced between the turbinate and septum, it straightens out by reason of its elasticity, forming a barrier from top to base of the septum. To obtain this result it is necessary to give the mica the shape which it should have in the nasal cavity where it is to be placed.

This substance, once placed in the nose, is so thin that after it is applied to the septum it is hard to distinguish from the latter. Further, as it does not in the least prevent escape of the secretions, and is not an irritating foreign body, cicatrization follows in the regular and normal way on each side. In these conditions, no matter what may be the mode of reaction of the patient's mucosa, the cut-off synechiæ have no tendency to reform.

I have employed the method in several very obstinate cases, after having removed adhesions by freely resecting the inferior turbinate. In spite of all the precautions taken and all my vigilance the adhesions formed again, often larger after each intervention.

I recall, in particular, the case of a young lady, operated upon for a septal spur, then a turbinectomy on the same side, in which the synechiæ were such that the corresponding nasal fossa was almost impermeable to air. By interposing a mica plate, after cutting the cicatricial bridges, I got perfect cicatrization and normal permeability. The patient was refractory to cocain, which in her produced a troublesome vasodilatation, so that it was almost impossible to obtain perfect anesthesia, even by adding a little adrenalin. Subsequently,

the turgescence of the mucosa, which set in at the beginning of the operation, never ceased during the whole period of cicatrization, making adhesion almost inevitable. This was a quite unfavorable case, in which chromic acid and various kinds of plates had always failed.

Because I know the inconvenience of these synechiæ and the difficulty of preventing their formation, I have thought it useful to my confreres to point out this new mode of treatment, which, I am certain, will in other hands than mine give similar results to those which I obtained.

REFERENCES.

1. Revue Hebdomadaire de Laryngologie, No. 17, p. 756.
2. The Laryngoscope, 1898, No. 1.

LXVI.

ANTISEPSIS RATHER THAN ASEPSIS IN THE TREATMENT OF OTITIS MEDIA PURULENTA.*

By Samuel Theobald, M. D.,

Baltimore.

It has long been a matter of astonishment to me that in the minds of otologists of experience there should be any doubt as to whether antiseptic agents are or are not of value in the treatment of acute or chronic otitis media purulenta. In my own mind, certainly, clinical observation has left no room for such doubt. My conviction as to their efficacy is so clear, indeed, that in employing boiled water or a sterile wick to control a suppurating ear, I should consider myself only less censurable than in treating a gonorrheal conjunctivitis in similar fashion. When, therefore, I read in a very recently published treatise devoted in great part to diseases of the ear, that in the treatment of otitis media purulenta the quantity of the fluid used, to insure a thorough washing away of all the pus, is more important than its chemical nature, and when I hear similar views advanced by otologists, as they often are, in formal and informal discussions, I wonder—well, I wonder if those who express such views have really made any comparative clinical observations of their own, or whether they have simply accepted someone else's supposedly authoritative dictum, or have merely been swayed by the fashion of the day.

Twenty-four years ago, when it was the custom to treat cases of acute as well as chronic suppurative otitis media by the insufflation, and even the packing, of powders—boracic acid especially—into the external auditory canal, I read a paper before this society[1] in which I advocated, as a safer and equally effective procedure, especially in acute purulent inflammation of the middle ear, the employment of boracic acid in saturated solution, and in support of my contention re-

---

*A paper read before the American Otological Society, June 11, 1912.

ported a number of cases treated successfully in this way. Needless to say, the discussion which the paper elicited was characterized by a considerable measure of skepticism.

Four years later I presented to this society another paper upon "The Value of Weak Solutions of Bichlorid of Mercury in the Treatment of Otitis Media Suppurativa,"[2] and reported several cases in which, after boracic acid in solution had proved ineffectual, a bichlorid solution, 1 to 8000, had promptly controlled the discharge and brought about a closure of the perforation in the tympanic membrane.

In the years which have elapsed since this paper was written, I have come to place chief reliance upon the mercuric solution, though cases have been met with occasionally in which the boracic acid solution has proved more efficacious. And it is upon the abundant experience of these twenty years that my faith in the value of antiseptics in the treatment of otitis media purulenta is based.

As to the method of employment, I may say that the boracic acid has been used in saturated solution, the bichlorid, at the outset of the treatment, in the strength of 1 to 8000, though not infrequently this has been increased to 1 to 4000. If the discharge is copious, the ear is thoroughly but gently syringed, or douched, with whichever solution is employed, and, even at the beginning of the treatment, it has seldom been found necessary to do this oftener than two or three times daily. When the discharge is scant the ear is cleansed with the cotton mop; if the acute stage of the attack has subsided sufficiently to make it permissible, the discharge in the tympanic cavity is driven out by the Valsalva method or by the Politzer bag; the ear is again wiped out, and a sufficient quantity of the warmed solution is poured into the ear to nearly fill the canal. Before this is done the patient is placed in such a position, with the head inclined, that the ten minutes, at least, during which the solution should remain in the ear shall not prove too irksome. The solution is then allowed to run out, the ear dried, not very thoroughly, and the orifice of the meatus closed with a soft plug of absorbent cotton. How frequently this application is made depends upon the effect produced, but it is never oftener than once in twenty-four hours.

I may add that I have felt no temptation to use the gauze wick that is now so much in vogue. If the discharge from

the ear is profuse the wick must unquestionably obstruct the outflow, unless it is changed more frequently than is possible except in hospital practice, with the services of a skilled assistant at command. While if it is slight, it is not apparent how the wick can be of appreciable advantage. At all events, when efficient antiseptics are used the need for it is not evident.

When, formerly, I employed boracic acid oftener than I do now, I found, as the case progressed towards recovery, that its too frequent use was detrimental, since, by keeping the parts too dry and the tympanic membrane too anemic, the healing of the perforation in the drum head was retarded or completely arrested; but this does not seem to happen when the bichlorid solution is used.

There are few cases, even of chronic otorrhea, that seem to me to call for other treatment—apart, of course, from operative procedures—than the employment of the bichlorid solution in one of the ways indicated; but exceptionally, especially when there is extensive destruction of the drum head, the insufflation of boracic acid or of boracic acid and aristol or oxid of zinc, equal parts, has been found efficacious when the mercuric solution has failed.

I could exhaust your patience by citing many cases from my clinical records in proof of the efficacy of the treatment I have outlined; but I shall content myself—and am sure of your approval in doing so—with a brief account of a single typical and very apposite case which recently came under my care.

A Baltimore lad, at school in New England, had an acute otitis media, for which he was sent to a specialist in Boston. The membrane was incised, and directions were given that the ear should be syringed with "water" (doubtless boiled water) only. After eight days of this treatment, as the discharge continued, he was brought home, and came at once to see me.

The canal was found to contain a considerable quantity of pus, and the drum head was so opaque and sodden as scarcely to be distinguishable. The pus was wiped out with a cotton mop, and bichlorid solution (1 to 8000) instilled, and allowed to remain in the ear for ten or twelve minutes. The ear was then dried and closed with a cotton plug, as has already been described. Syrup of the phosphates of iron, quinin and

strychnia, in dram doses, was prescribed. The next day the ear was clean and dry, and the appearance of the membrane had changed greatly for the better. The bichlorid instillation was repeated, and the patient was directed to resume the use of a nasal spray—bichlorid of mercury in normal salt solution, 1 to 5000—which I had prescribed for him several months previously. On the following day the drum head was beginning to present a fairly normal appearance, though still hyperemic, and Valsalva inflation gave a distinct, dry, nonperforation sound, after which hearing for whispered words was found to be practically normal. As a matter of precaution a final instillation of the mercuric solution was made.

Two days after this,[a] though the membrane was less hyperemic and hearing for the voice normal, he reported having had some twinges of pain in the ear since his previous visit. For this the solution of the alkaloids of atropin and cocain in oil of sweet almonds,[3] which I suggested many years since as a useful remedy in the early stages of otitis media, was prescribed, eight drops in the ear, three times a day.

Two days later, there having been no recurrence of pain, the ear was in such "good shape" that the patient was given permission to return to school.

Could there be a more striking refutation than this case affords of the teaching, so prevalent at the present day, that it is the quantity of the fluid that is thrown into the ear, not its chemical composition, that is of importance?

As a final observation, I may add that since the great majority of cases of acute otitis media, if not purulent from the outset, become so, in spite of aseptic precautions, after the membrane is incised or a spontaneous rupture has occurred, it is, in my opinion, wise, in all cases, to begin the antiseptic treatment without loss of time or waiting for further developments.

REFERENCES.

1. Trans. American Otological Society, 1888.
2. Trans. American Otological Society, 1892.
3. The formula is: Atropin (alk.), gr. j; Cocain (alk.), gr. ij; Ol. amyg. dulc., dr. ij.

OTITIC EXTRADURAL ABSCESS AND SINUS THROMBOSIS. REPORT OF A CASE WITH REMARKS.*

By James F. McCaw, M. D.,

Watertown, New York.

The case which forms the subject of this report was that of a male, 29 years of age, referred to me on March 16, 1912. The following history was elicited: About one week before coming under observation he contracted a head cold, which was followed in three days by severe earache in the right ear. This has continued ever since with increasing severity, so that he was unable to sleep for several nights. There has been no discharge and no swelling about the ear, but severe pain in and around it. Upon examination the canal was clear, the membrana tympani deeply congested, with necrosis of the superficial epithelium. There was marked bulging of the membrana flaccida and Schrapnell's membrane; hearing was nil for the watch. No apparent swelling over the mastoid, but slight tenderness to deep pressure over almost the entire area; no point of special tenderness could be determined at this time. Physical condition excellent. The patient being from out of the city, was sent to the hospital, and a free myringotomy done. This gave him immediate relief from his suffering, and a profuse, purulent discharge appeared, with the disappearance of all mastoid tenderness. The discharge gradually diminished and in ten days was very scant. The condition of the middle ear showed proportionate improvement, so the patient was allowed to return home with instructions for the care of the ear and to report in one week.

Upon examination one week later there was a profuse purulent discharge from the opening in the tympanic mem-

brane, and there was sagging of Schrapnell's membrane and
the posterior superior canal wall; some tenderness over the
entire mastoid area, but now there was extreme point tender
ness over the region of exit of the emissary vein; tempera
ture, 99°; pulse, 86. Said he felt perfectly well except for
the ear discharge.

In view of the clinical manifestations, I insisted upon his
going to the hospital for a mastoid operation. This was done
the next morning, and on uncovering the mastoid, pus was
not encountered until reaching the deep cells, where it was
found the infection was expending its force. The bony wall
overlying the sigmoid portion of the lateral sinus was necrotic
throughout its entire length. This was cut away and just
above the knee of the sigmoid an epidural abscess was en-
countered and about a half dram of pus evacuated; no gran
ulations were present on the vessel wall. This abscess had
exerted so much pressure on the sinus wall that a clearly de
fined pit was left after evacuation. The sinus below this point
was round, firm and resistant, without pulsation. At this
stage of the operation, in view of my unsuspected findings, it
was a question in my mind just what course to pursue. As
this patient had given no clinical symptoms of thrombosis of the
lateral sinus, I determined to clean the area as thoroughly
as possible, pack it as usual and watch developments. This
was done. Examination of the pus showed a streptococcus
infection. A differential blood count was made every two
or three days for nearly three weeks. This patient went on
to a complete and uninterrupted recovery. The only un-
usual thing noticed in the healing process was that the sinus
wall at the upper part did not cover over until the other part
of the wound was well filled with granulations.

There are several points of interest to me in this case. The
first to impress itself is the impossibility of making a diag-
nosis of sinus thrombosis before opening the mastoid, as there
was absolutely no clinical evidence of sinus involvement, and
not until the sigmoid groove was reached and the wall found
exposed from necrosis of its bony covering, was it even sus-
pected. Although the findings showed a very extensive in-
fective process around the sinus with the accumulation of a
fair sized epidural abscess which had made sufficient pressure

on the sinus wall to leave a pit in it, even after evacuation, there were no clinical symptoms of absorption. This, to the writer, seems rather unusual. Again, was this an infective or noninfective thrombus? This question I am at a loss to answer.

Whiting, in his most excellent monograph on sinus thrombosis, in speaking of the first stage, says: "In this stage recovery is still possible, although improbable without operation upon the sinus, the infective process occasionally resulting in a constructive inflammation, terminating in cicatricial obliteration of the sinus lumen, a conclusion fervently to be desired, but very seldom realized. The anticipation of such a favorable outcome is to be entertained only under most exceptional circumstances, namely, when the virulence of the infection is so far attenuated as to have nearly expended its energies during the invasion of the vessel walls and in the production of the resulting clot, and when the residual activity of the pathogenic organisms present in the thrombus is speedily destroyed by the germicidal action of the phagocytes and leucocytes. Under no circumstances can such thrombi be regarded as noninfective; that the extension to the sinus of an infective inflammation and the introduction into it of infective germs, should produce a noninfective clot, would be an anomalous state of affairs. That, however, the infection may be sufficiently attenuated after producing the thrombus to fail of causing disintegration of the same, in view of the action of the phagocytes and leucocytes, can be readily comprehended." On the other hand, Phillips, in his recent work on Diseases of the Ear, Nose and Throat, takes the opposite position and says: "In the absence of the classical symptoms of infection or thrombosis of the sinus prior to operation, even though a perisinus abscess is discovered, it is inadvisable to explore it either by incision or by puncture, unless its walls are necrotic or gangrenous. Even if the surgeon is convinced that a clot is present, if no symptoms of infective thrombosis have appeared, it is inadvisable to interfere surgically with the sinus. The author is firmly convinced that noninfective thrombi may develop in the lumen of a venous sinus which eventually become organized into connective tissue. To operate upon cases of this type and thereby brave the danger

of infecting the sterile thrombus, is a questionable procedure."

The writer is not prepared to take issue with either of these authors as to the possibility of a noninfective thrombus in the presence of an infective area about it, but we do think that the patient's interest is subserved by not attacking the sinus surgically where there is an absence of clinical symptoms pointing to septic absorption; there can certainly be no harm in waiting until such evidence is present. Under close observation this can be detected at its inception, and the patient's welfare will be only slightly, if at all, jeopardized, with the possibility of an uninterrupted convalescence, without further surgical interference. Frequent blood counts will render valuable assistance, not only in showing us the amount of absorption taking place, but is also a valuable aid in indicating the resistance of the individual. I am now having such examinations made in most of my cases, and am coming to place more and more reliance upon the findings. The outcome of my case at least warrants the procedure adopted.

LXVIII.

HYSTERIA SIMULATING MASTOIDITIS, WITH SUSPECTED BRAIN ABSCESS FORMATION.*

By S. MacCuen Smith, M. D.,

Philadelphia.

Patient, H. T., colored, aged 26 years. Occupation, porter. Seven months prior to his appearance at the Jefferson Hospital clinic, the patient was hit by a baseball over the mastoid process on the right side, the blow being followed by bleeding from the right ear and unconsciousness for twenty-four hours, after which he was totally deaf in the right ear, and so deaf in the left that one had to speak in a loud tone, close to the ear, to make him hear conversation. The hearing in the left ear, however, was restored within a short time.

There was excruciating pain over the right mastoid process, extending from the occipital to the parietal region, and continuing more or less severe during the entire seven months. After the accident the patient suffered from constant buzzing tinnitus aurium and throbbing pain in the right ear. He also had periodic epistaxis, increased when lying down, and frequent attacks of vertigo, so severe that to prevent falling he was obliged to assume the recumbent position. He never had any attacks of unconsciousness after the one immediately following the accident. The patient also suffered from complete left hemianesthesia, in proof of which a pin could be run into his flesh at any point on that side without apparently causing pain.

Examination showed the ear to be practically normal in all respects, save the apparent severe pain over the mastoid process and temporal region, this pain being markedly increased by even slight pressure. The external auditory canal, membrana tympani and ossicles, in so far as could be

---

determined by a physical examination, were normal. In the right ear there was complete absence of both aerial and bone conduction, and we were unable, during frequent and varied tests, to change this conclusion.

Blood examination. This report shows a moderate leucocytosis (14,400), with an increase in polymorphonuclears (84 per cent).

Ophthalmologic examination. The report shows the vision in the right eye 20/50 plus, the right nerve head markedly hyperemic, and the physiologic cup filled in. The rest of the fundus is normal. There is a simple hyperopic astigmatism of 1.00 D, which no doubt accounts for his poor vision. The vision in the left eye is also 20/50, the nerve head is similarly hyperemic and the cup filled in. There is no gross lesion. Simple hyperopic astigmatism, 1.00 D. The hyperemic nerve head stands out in marked contrast to the surrounding fundus, which is typically negroid.

X-ray report. Mastoid cells are of small area, but the individual cells are large and there is some bone defect in the upper portion. The process is in no part sclerotic. The lateral sinus is somewhat superficial. There is no positive indication of an intracellular exudate, but the shadow is suggestive of this condition.

Neurologic examination, by Dr. Geo. E. Price, shows gait normal, with eyes open; slight ataxia with eyes closed. Station, somewhat plus sway. Ataxia of upper extremities questionable. Pupils equal and normal in reaction; no nystagmus nor external ophthalmoplegia. When first seen, the musculature of the face was apparently normal; when examined the following day, there was slight drooping of the mouth on the left side; muscular movement normal, however. Movements of upper extremities normal. Abdominal and cremasteric reflexes present. Left leg weak, as compared with the right leg. Knee jerks plus on both sides. No ankle clonus on either side, although a pseudoclonus was at times obtained. Babinski's sign absent. There is a diminution of sensation over the entire left half of the body.

While hysteria was discussed as a probability, the fact that the ear symptoms were in one side and the sensory and motor symptoms on the opposite side, together with the blood findings, led to a diagnosis of abscess of the right temporal lobe.

The temperature and pulse, both preoperative and post-operative, were practically normal. This, of course, would not bear out our suspicion of brain abscess formation, and yet, when we consider how frequently the classical pressure symptoms, as indicated by lowered pulse, respiration and temperature. fail in the presence of brain abscess, we were probably justified in suspecting that condition, after a careful consideration of the history of trauma, the presence of hemi-anesthesia, the eye report, the nervous phenomena present, and the blood picture.

Five days after the patient was admitted to the hospital a simple mastoid operation on the right ear was done for exploratory purposes. After the removal of a very hard cortex no actual disease was discovered in any part of the cavity, though the tip of the mastoid bled more than normal. One day after the operation the hemianesthesia cleared up, the vertigo disappeared, and in due time the hearing became nearly normal in the right ear, having been previously regained in the left, as above stated.

This patient was seen quite recently, or about four months after the operation, and the improvement incident thereto is still maintained, which clearly shows that we were wholly deceived by a typical neurasthenic, and although the patient might have recovered under nonsurgical psychotherapeutic treatment, as was probably indicated by the preponderance of subjective over objective symptoms, yet we felt justified in performing an exploratory operation for the reasons above stated, and ordinarily, if the patient had suffered from an otitis media, these symptoms, taken together, would offer a complete picture for operative intervention for mastoid disease. and the findings at the time of operation would have enabled us better to determine the presence or otherwise of an intracranial lesion.

LXIX.

SUDDEN, TOTAL, AND PERMANENT DEAFNESS IN ONE EAR, FROM SUPPOSED LABYRINTHINE EFFUSION, FOLLOWED FOUR YEARS LATER BY SIMILAR CONDITIONS IN THE OTHER EAR.*

By James A. Spalding, M. D.,

Portland, Maine.

Puzzling and interesting as are many diseases of the ear, none is more so than those enigmatic attacks of deafness supposed to be due to a labyrinthine affection, but which, in the absence of a postmortem examination, we can never precisely define. Moreover, the rarity of such attacks adds to their otologic interest, so that it becomes a scientific duty for every physician who observes them to report their details for the purpose of adding to our slight information concerning primary diseases of the inner ear.

A woman of fifty-five, with perfect hearing in both ears, was operated upon some ten years ago for suspicious glandular enlargements in the axillæ, and made a perfect recovery, so that at this date she presents no possible symptoms of sarcomatous disease. The patient had also been troubled previous to this operation, and suffered long afterwards, intercurrent with the aural affection about to be described, from numerous attacks of typical episcleritis, supposed to be of rheumatic or gouty origin. At irregular intervals one eye or the other, or both, would become involved, the sclera taking on a pinkish and rapidly a violet color, accompanied with pain and lacrimation, so that the eyes could not be used at all for near work. Nothing proved of any avail for a cure, though the conditions were relieved and shortened by cocain and hot packs. Fortunately for the patient, the inflammation re-

---

mained limited to the sclera, never invaded the cornea or iris, and she can still see in the distance and near to with properly fitted spherocylindrical ·lenses: $+$ 2. D. S. $\subset$ $+$ 0.50 D. C. axis 90° O. U. for far, and $+$ 5, with cylinders as above, for near.

The patient also suffered during all those years from rheumatic or gouty pain, tenderness and swelling in the joints from time to time, and found relief from using the salicylates. She had no arteriosclerosis.

The patient was attacked in 1902 with double pneumonia, was at one time very low, but gradually rallied and during her convalescence developed the following aural condition: She was resting on her right side with her right ear imbedded in the pillow and listening to the conversation of a friend, when she suddenly perceived in her left ear an intense buzzing sensation, then instantaneous loss of hearing, and exclaiming: "I cannot hear a word that you are saying," she raised her head from the pillow, but fell back at once on account of vertigo and nausea. Dreading to move again, but anxious to hear what could be done for her relief, she was turned on her right side, and with her right ear was now able to hear as well as ever. A few days later she could be lifted to a chair, and gradually was able to walk with assistance, but the vertigo persisted, so that at the least attempt to walk she fell toward the right.

Examination of the left ear showed no abnormality, the drum head, light spot, mobility and transparency being normal. There was no visible hyperemia of the promontory. The tube was normal and easily inflatable. So far as could be determined the ear was totally deaf to voice, watch, and tuning forks, although, owing to the intense tinnitus, and the defective bone conduction at the patient's age, satisfactory replies could not always be obtained. Tests with Galton's whistle also showed defective hearing throughout its entire compass.

Although the patient slowly recovered strength after her rally from the pneumonia, and made determined efforts to move about, she could not walk alone, and gradually fell into the habit of pushing out her left foot first, in trying to walk when assisted.

For a long time she was obliged to rely upon others upon

the stairs, but gradually was enabled to do part of the task alone, with the aid of the banisters. She can in these days climb more easily and more securely than she can descend the stairs, but cannot even yet trust herself without an attendant. Tinnitus persisted; it was of the same disagreeable buzzing character, in spite of all treatment that could be borne. Nothing could be attempted with powerful remedies like thiosinamin or pilocarpin, because anything involving the least risk to the general bodily condition was absolutely negatived by the family physician. He opposed any experiments, and very properly, too, considering their problematic benefits. I may, however, say, in passing, that pilocarpin has been known to increase tinnitus and weaken the heart, while thiosinamin, although of benefit, exhibited in capsules or hypodermatically in the form of fibrolysin, might in this patient have excited irritation in the cicatricial remains of the former axillary operations.

The middle ear was inflated and atomized, hot air was driven into the tympanum through the catheter and directly against the membrana tympani through the external meatus, vibratory massage was applied to the mastoid, Lucae's pressure probe was used on the short process of the hammer, and Siegle's speculum was employed for manipulating the ossicles and preventing possible anchylosis. The ear was thus treated for months, but no hearing obtained. If, occasionally, the voice seemed to be perceived, the perception ultimately resolved itself into mere lip reading. Possibly in this test the other ear was not totally occluded, something very difficult to do in testing unilateral deafness. After the use of all the remedies that experience or the authorities could suggest, treatment was abandoned as hopeless.

Four years later, almost to a day, the patient then being in perfect health, the right ear was affected precisely as the left had been. The patient was talking with her attendant, when suddenly she ceased to hear. A slight sensation of vertigo accompanied the loss of hearing, and a few moments later, on trying to move, the tendency toward the right, which had so long been obsedved, changed into one of falling forward. Instead of pushing out her left foot, as of old, both feet participated in a movement like trying to push both feet into loose slippers lying on the floor. On the same day the patient's

voice, which had remained normal, suddenly changed into a sort of childish whimper, starting for a word or two on the normal pitch, then rising quickly into a high falsetto, where it has mostly remained ever since, though of late sounding more normally.

Examination of the ear revealed nothing abnormal. A consultation brought out the diagnosis of labyrinthine hemorrhage, with suggestions of treatment with galvanism and vocal massage. Galvanism was used persistently for months, with a weak current and with various changes of currents, but without effecting any improvement in the hearing. Thiosinamin and ergot were used a few times, hypodermatically, but also without effect.

Speech exercises and vocal massage according to Urbantschitsch were at that time much in vogue, and with unremitting intelligence the attendant tried all sorts of words and sentences, first into the ear first affected and then into the other, but both remained without improvement. The apparatus employed was an ordinary roll of cardboard with a bit of absorbent cotton passed in part way, to prevent too loud sounds impinging upon the drumhead.

Maloney's otophone and the acousticon were likewise employed with the same end in view, but effected no improvement. I may say here that Maloney's apparatus remains an instrument safe and valuable, while I am timid of the other, believing that all such resonators may endanger the hearing by too great a volume of sound, to say nothing of the greater effort demanded to perceive with them as the battery loses its power.

All machines for assisting the deaf to hear are based on the false theory that the hearing can be increased by conducting loud sound into the ear. This is as tenable as to claim that sight can be increased by throwing more light into the eyes. Loud sounds may also injure the hearing, for as they may injure the normal ear despite the natural mufflers of noise (the ossicles and membranes of the labyrinthine windows), so they may still further injure the diseased ear which has lost its mufflers by anchylosis or otherwise.

It is to be regretted that no otologist, so far as I am aware, has yet written intelligently concerning the real value of all these machines. I had even hoped to find them mentioned

for discussion here. Surely they demand proper investigation, when we consider the sensational claims made concerning their value to the ear, and the enormous sums demanded for them in comparison with the results abundantly demonstrated upon defective eyes by lenses, at very moderate prices.

To complete my story. The patient in these days, ten years later, carries on conversation by means of pen and pencil and lip reading. In the latter, however, she does not make so much progress as if she had begun when young. The entire sound conducting apparatus seems normal. The voice is natural. She can now walk fairly well without assistance. She hears nothing.

REMARKS.

Although the textbooks devote considerable space to labyrinthine diseases, most of it is given to suppurative conditions, either idiopathic or as an extension of middle ear suppuration. Much is also said of Meniére's symptoms, and of conditions dependent upon toxic labyrinthitis due to fevers or meningitis. But primary diseases of the labyrinth are rare. In addition to the one here reported, I can recall from 5000 patients only one more, which was unilateral, and occurred after long-continued organ playing. No improvement was obtained, but the vertigo and tinnitus ceased. The patient, staggering at first, was later able to walk with perfect equilibrium. I have also seen one other, in which, however, extension of suppuration could not positively be excluded.

Much stress is laid in the textbook on tests with tuning fork in labyrinthine affections. Although they proved negative in my patient, something concerning them may here be said. Such tests are, in my opinion, of little value, because most people have no idea of tones. Many musicians, even, cannot "place" certain forks in the octaves to which they belong. Many persons cannot tell a sound from a tone. Even with a hearing apparatus it is difficult for me to decide whether what I hear is a tone or the blow of the hammer upon the piano strings. 'Assuming that people with less musical education than my own have doubts like myself, I can say that many answers from patients are due to self-deception or misunderstanding of what is asked of them. Many patients assert, for instance, that they perceive a tuning fork best in

their better ear by bone conduction, simply because they know that it is their better ear, and therefore they must hear best with it. Yet we know that forks should be perceived loudest in the worse ear, according to the laws of obstructed conduction.

Age also interferes greatly with tuning fork tests. The older the patient the less the chances of obtaining any results at all toward diagnosticating labyrinthine disturbances. A good deal is also said about the exclusion of overtones, but this is all gratuitous, because it is as much as patients afflicted with deafness can do to perceive any tones at all.

Furthermore, anybody can assure himself with a Galton whistle that most people with the universal lack of observation insist on hearing the noise made by the wind issuing from the tube, instead of the actual shrill note, and observers are consequently misled regarding what is actually heard.

CAUSATION.

The only suggestion offered regarding the origin of this condition, as here related, was a labyrinthine hemorrhage. With this I at one time coincided, but afterward I came to a different conclusion.

Here is a patient without heart disease or atheroma, who loses the hearing in one ear during convalescence from pneumonia. Four years later, without any intercurrent disease, her heart still healthy and her arteries free from atheroma, she loses the hearing in the other. Is it reasonable that so minute a hemorrhage as would occupy the labyrinth could occur, first in one ear and then in the other, after so long an interval? If we recall the numerous attacks of episcleritis and of effusions in the joints, and if we remember the number of women who suffer at about the age of my patient with iritis serosa or episcleritis, with more or less loss of vision, is it not rather more reasonable to imagine in the present instance a serous or leucocytic effusion into the labyrinth, than one that was hemorrhagic? In the absence of a necropsy we have no proof of our belief, but just as in iritis serosa we have an invasion of the optic nerve sheath and uveal tract with small cells, and in episcleritis a leucocytic invasion of the anterior portion of the eye, why may we not have cellular

or leucocytic invasion of the labyrinth, as a whole, from the same causes that in so many women produce the infiltration of the organ of sight? In point of fact, many labyrinthine sections already published exhibit such cellular and leucocytic infiltrations. However, this is all mere speculation, but is worthy of deeper study.

Voss* mentions a case of serous labyrinthitis, but does not prove it beyond a reasonable doubt. Bárány also is mentioning similar cases in this congress.

If a pneumonia produced the deafness in the first ear, we have nothing like that to account for the attack in the second ear. Nor does my case resemble a genuine Meniére, with its lack of vomiting, repetitions, impairment of handwriting, or loss of consciousness.

A nurse whom I observed for several years suffered many times from unilateral rheumatic optic neuritis with loss of sight, infiltration of the nerve sheath. A cure was effected by the use of salicylates.

Acute auditory nerve neuritis has also been mentioned as due to rheumatism.

If the deafness in the first ear were due to a thrombus or embolus, it would be extraordinary that without any cardiac affection during four years the second ear should become deaf from the same cause.

TREATMENT.

All remedies that my experience or those of the authorities or consultants suggested were tried without benefit. Forcible measures, like long-continued hypodermatic injections of pilocarpin, fibrolysin or ergot might have been of avail, but were contraindicated in a delicate patient. Lumbar puncture might be tried in a similar case, but was not in mine.

Cheatle long ago claimed that the endolymph and the perilymph were an essential portion of the sound conducting apparatus of the ear, and in no physiologic sense could be claimed as belonging to the sound perceiving apparatus, but his views do not seem to have attracted much attention. Based on this idea, he suggested that in cases more or less resembling this present one, surgical puncture of the outer labyrinthine wall would be good and conservative surgery.

*Knapp's Archives of Otology, XXXVII, p. 391.

From the point of view of an oculist, this proposal reminds us of opening the sheath of the optic nerve in optic neuritis with the intention of evacuating effusions in that locality.

Careful study of otologic literature has failed to reveal to me a single case of primary labyrinthine disease in the least resembling the one which I have had the honor to report. As others of this nature may have been observed, yet have never been reported, I hope that the material now offered for consideration may induce my confreres to mention instances in their experience which they have not published because they had never been able to substantiate their opinions by the results of a postmortem examination.

SYMPTOMS OF THROMBOSIS OF THE LATERAL SINUS—REPORT OF A CASE IN WHICH AN EXACERBATION OF VISCERAL LUES SIMULATED THE SYMPTOMS OF LATERAL SINUS THROMBOSIS.

By Jesse W. Downey, Jr., M. D.,

Baltimore.

Following an operative procedure for the cure of a suppurative condition of the mastoid process and tympanum, a sudden chill and an ensuing marked rise in temperature strongly indicate a beginning general septicemia due to septic thrombosis of the lateral sinus. Having excluded lobar pneumonia and malaria as possible causes for the initial rigor and fever, and the mastoid process being the only definitely known focus of infection, an irregular febrile temperature, the curve indicative of sepsis, can usually mean but one thing; and when accompanied by frequent chills and debilitating sweats, the diagnosis of a crumbling thrombus of the lateral sinus is absolute. The typical case presents other symptoms: constant headache, mental depression or delirium, tenderness over the mastoid emissary vein and in the posterior cervical triangle or along the course of the internal jugular, edema in the soft tissues of the neck and, occasionally, optic neuritis. All these symptoms may be absent. They are seldom all present until the stage of embolic metastasis. By the temperature curve alone, therefore, an operation may be vitally indicated.

In atypical cases the laboratory findings, when positive, are of confirmatory value; when negative, their exclusive evidence is not so trustworthy.

In the majority of cases of sinus thrombosis the streptococcus is the infecting organism; infection from other organisms is not, however, uncommon.

A leucocytosis with an increased percentage of polymor-

phonuclear cells is certainly to be expected; frequent reports upon the blood examination in these cases, however, prove that thrombosis may exist without a characteristic blood picture. In this respect it is well to remember, however, that purulent conditions of the mastoid process and surrounding structures do not, as a rule, occasion a marked increase in the leucocytes, therefore, a count showing ten to twelve thousand cells (looked on by the laboratory man as immaterial), with a relative increase in the polymorphonuclear neutrophiles, may be of diagnostic value to the otologist.

To Libman[1] belongs the credit of demonstrating the value of blood cultures in the diagnosis of sinus thrombosis. A bacteremia is very strong conclusive evidence of a diseased lateral sinus. All efforts to obtain a growth of organisms from the blood may fail, however, even in the presence of a thrombus, the existence of which is later proved by operative exposure. And that bacteria may be obtained from the blood very occasionally, the lateral sinus being normal and the mastoid alone involved, has been shown by Sondern.[2]

An early diagnosis in all cases of lateral sinus thrombosis is essential. Fortunately the majority of the cases seen, even when symptomatically atypical, are not obscure enough to delay the surgical treatment necessary. In the presence of mastoiditis, the occurrence of chills, a fluctuating temperature and sweats may be considered the cardinal symptoms of infection of the lateral sinus; all other signs may be absent or obscure, generally speaking, therefore, as one author has said, "to postpone operating is no more justifiable than it would be to withhold a rope from a drowning man with the hope that a wave might wash him ashore."

The following case is reported as a very rare instance in which the symptoms of a vague coexisting disease, occurring during convalescence from an operation upon the mastoid process, simulated to a confusing degree the temperature course characteristic of thrombosis of the lateral sinus. Except from the standpoint of differential diagnosis the case is of much more interest to the internist than to the otologist.

W. A., colored, male, age thirty-six, applied at the Ear Clinic of the Johns Hopkins Hospital Dispensary for treatment and was referred and admitted, under my charge, to the Baltimore Eye, Ear and Throat Hospital for operation.

History.—Four years ago a large hard lump had appeared under the jaw on the right side. This disappeared without treatment, but was followed by an ulcer on the side of his face near the external auditory meatus. He was told that this was "skin cancer" and was given X-ray treatment in Richmond, Virginia. The ulcer healed after several months of treatment, but the canal of the ear closed. During the time of the X-ray treatments the ear began to discharge. This discharge had persisted, and on account of the closure of the canal, and in view of the fact that he suffered a great deal of pain at times, his physician had advised him to come to Baltimore for further examination and an operation if necessary.

Examination.—In front of the right ear and extending to the angle of the jaw was a round white scar; the tragus had been destroyed either by the ulcer or an X-ray burn, and the external auditory canal was closed by fibrous tissue through which there extended a small fistula admitting a medium sized silver probe. There was a profuse discharge of creamy pus from the fistula, the flow being increased by pressure in front of the ear. Slight tenderness on deep pressure over the antrum of the mastoid. Temperature normal.

I advised an operation, with an enlargement of the canal and an effort to cure the suppuration of the middle ear as its object.

Operation.—After the usual preparation, a mastoidotympanal exenteration was performed; the bone was sclerosed and the mastoid antrum contained a small amount of granulation tissue. The tympanum was filled with fibrous granulations. A continuation of the same tissue was found in the floor and anterior wall of the external auditory canal. The ossicles had been completely destroyed. I made a larger tongue-shaped flap than usual, carrying the incisions well back into the concha. After removing the cartilage from the flap the layer of skin remaining was tightly packed back into the exenterated mastoid; the result was a large canal, the floor and anterior wall of which, unfortunately, was denude of skin on account of the inflammatory destruction.

The man was returned to bed in good condition. On the following day at twelve noon he had a slight chill, and his temperature, which had been normal, jumped to 102° F. At twelve midnight his temperature had returned to normal. The

next day the same thing occurred, namely, a sudden rise of temperature to 102° F. with subsidence to normal within twelve hours. The wound was dressed and found in fair condition. There was a free discharge of pus from the middle ear, and an ulcerative area on the floor of the canal.

From then on until June 15th, seventeen days after operation, the course was uneventful. The posterior wound healed by first intention, the purulent discharge persisted and, as was feared at the time of operation, the shrinkage and the formation of scar tissue in the floor and anterior wall of the canal were causing an unavoidable diminution in its size. On June 15th the man had a severe chill with a rise of temperature to 102° F. He had been up and about for ten days or more, feel-
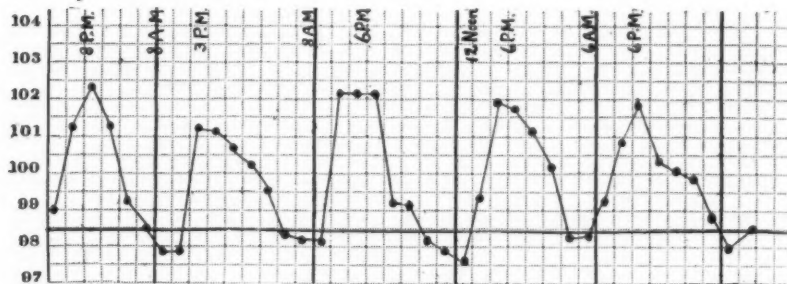
CHART NO. I.

The temperature curve here shown is characteristic of the febrile course during the two weeks, and more.

ing perfectly well, and had no complaint to make following the chill except that he felt languid. By morning of June 16th his temperature was normal, but by noon another chill had occurred followed by fever. From June 15th, as noted, until July 3rd, there was a rise in temperature, varying from 100° F. to 104° F. in each twenty-four hours, the rigor occurring shortly after noon, the maximal temperature being reached between three and six p. m., followed by a steady decline to normal or slightly subnormal by nine o'clock of the next morning. (See Chart No. I.) During the fastigium the patient's only complaint was that he felt weak. Sweating frequently occurred.

In view of the fact that the purulent discharge from the ear was still copious and, in spite of all preventive efforts, was being retained and dammed back into the mastoid by the closure of the auditory canal, a necrosis of the bony wall of the lateral sinus was possible, with the appearance of the febrile symptoms, it appeared highly probable that such was the case and that a septic thrombosis of the vessel had occurred. The lungs were normal and no malarial parasites could be found. Quinin was later administered without effect. The leucocytes were normal, namely, 6,140; the differential count showed no relative increase in the polymorphonuclear neutrophiles; the percentage of mononuclear cells was rather high, 23.45 per cent, of which 15.54 per cent were small mononuclears and 7.91 per cent large. Such a blood picture seemed to exclude sepsis.

The man's general condition being so good, to keep him under observation seemed permissible, therefore, for the next week a careful study was made of his case. Frequently made leucocyte counts were always normal, the highest count recorded being 6,880. Blood cultures made on two occasions were negative. In the absence of all other symptoms the temperature curve would have been sufficiently convincing of involvement of the lateral sinus, had it not been that the temperature invariably dropped to normal or subnormal in each twenty-four hours.

To Doctors Roger S. Morris and J. W. Churchman of the Johns Hopkins Hospital I am indebted for the correct diagnosis. A complete physical examination made by them disclosed nothing of interest except two scars, one upon the shoulder, the other on the thigh, similar in appearance to the face scar. The man admitted luetic infection, the initial lesion having occurred eight years ago. . The scars were suggestive, when taken in connection with the high percentage of mononuclear cells present in the blood, of the present condition being due to an obscure tertiary lesion. A Wassermann test, made at this time, gave a triple positive reaction. Search was made for the spirocheta pallida in the discharge from the ear, but without result, the infection being a mixed one, staphylococci predominating.

An intravenous injection of salvarsan was given July 3rd, which was followed by a marked reaction with a jump in the temperature to 104° F., after which the temperature fell rapidly to normal and remained so. (See Chart No. II.)

In a paper entitled "The Strauss Test for Hepatic Insufficiency" Dr. J. W. Churchman[3] has included the case in a series reported to show the value of the Strauss test. I quote his report in part:

"There were suggestive scars on the shoulders and legs, and the Wassermann reaction was found to be strongly positive. A probable diagnosis of visceral lues was made, the case resembling those reported by Mannaberg and others. The liver was very slightly enlarged, on percussion; palpation was of no help on account of abdominal rigidity. Specific treatment was instituted, beginning with salvarsan. The temperature promptly dropped to normal (see chart) and has remained so ever since (nearly three months later). The Strauss test, made before treatment was started, showed a very marked alimentary levulosuria; the Seliwanoff reaction,
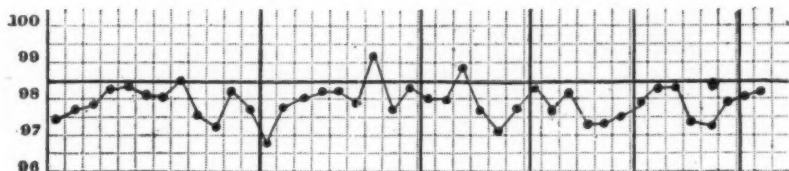
CHART NO. II.

Temperature course immediately following the use of salvarsan.

five hours after ingestion, was stronger than in any other case in the series, and remained strongly positive six hours after ingestion.

Results of the Strauss test, July 1, 1911:

	S	B	PH
1	0	....	....
2	0	....	....
3	++	....	....
4	++	....	+
5	+++	....	....
6	+	....	+

After the patient had been on treatment for a little over two months and had been running a normal temperature for this length of time, the Strauss test was repeated. The Seliwanoff reaction was negative in all the specimens, as was the Fehling

reaction; and, though sugar was shown to be present in the second, third and fourth specimens, by the Nylander and phenylhydrazine tests, the very marked difference from the reaction of two months previously was most striking.

Results of the Strauss test, September 21, 1911:

	S	B	F	N	PH
1	?	....	0	0	0
2	0	Trace	0	+	+
3	0	"	0	+	+
4	0	"	?*	+	+
5	0	"	0	?	0

Visceral lues was probably present in this case. Conclusive evidence that the liver was involved is wanting; but the clinical similarity of the case to the reported cases of hepatic lues suggests this possibility. The complete cure by specific treatment and the simultaneous disappearance of a positive alimentary levulosuria are the interesting features."

In conclusion, I wish to say that I again operated on the man and by the use of Thiersch skin grafts fully succeeded in closing the fistula and stopping all suppuration.

REFERENCES.

1. Libman and Cellar: The Importance of Blood Cultures in the Study of Infections of Otitic Origin. American Journal of Medical Science, Vol. CXXXVIII, p. 409.

2. Sondern, F. E.: Blood Cultures in Otology. Annals of Otology, Rhinology and Laryngology, Vol. XX, No. 3, p. 621.

3. Churchman, J. W.: Johns Hopkins Hospital Bulletin, January, 1912.

---

*No true reduction; greenish precipitate on standing.

LXXI.

CASE OF BILATERAL INFLAMMATION OF THE EXTERNAL AUDITORY CANAL FOLLOWED BY BILATERAL SINUS THROMBOSIS—AUTOPSY.

By Dunbar Roy, A. B., M. D.,

Atlanta.

During the last few years a great deal has been written upon the subject of sinus thrombosis in its relation to a diseased condition of the middle and internal ear, but so far as I have been able to ascertain there is no reference in the literature of ear diseases to the question of sinus thrombosis following any diseased condition of the external ear. All text books treat very light the prognosis of furunculosis of the external auditory canal, and up to the time of observing this case I myself considered furunculosis in this locality by no means a dangerous disease, for with the exception of the intense pain at the time of the acute process, no complications had even been seen to occur, nor the final hearing in the least diminished.

Case History.—Miss M., about 50 years old, was a strong, robust woman, the matron for a large orphans' home. Had always enjoyed the best of health. Had never suffered from any previous aural disease.

March 8, 1911, patient consulted me at my office on account of severe pain in the right ear, which had been present for two days. Examination showed the right auditory canal to be very much swollen and very sensitive to the slightest pressure. No point of fluctuation found, but a diffused inflammation of the whole canal. A purgative was ordered, with a local tamponade of absolute alcohol and hot fomentations.

March 10th. Patient returned with comparatively no improvement. Pain quite severe. A deep incision was made on lower anterior wall of the canal. Free hemorrhage, but no pus.

March 11th. Patient feels much better. Quite a discharge of pus. From this time on the symptoms gradually subsided, pain disappeared together with the swelling in the canal, so that the drum membrane could be distinctly seen.

March 15th. Patient returned with almost identical conditions in the left auditory canal as had existed in the right. Parts swollen and tender. Temperature normal, but patient complains of headache. Same treatment as previous. Free incision brought only free bleeding.

March 16th. Patient has been suffering considerable pain. Another free incision was made into the canal, and hot irrigations ordered. Codein was given at night.

March 17th. Patient slightly relieved. Some discharge of pus, and the swelling diminished. Following this visit the ear became quiet and most of the tenderness disappeared. The patient continued her work without interruption, and I did not see her again until April 3rd, two weeks later. The patient then complained of headache, mostly on the right side, and reported that the same had been present almost continually. Examination showed the right ear to be almost normal in appearance. In the left there was still some swelling in the canal, but no tenderness. The drum membrane had only the faintest marks of congestion. I could see nothing in the condition of the ears to cause the intense headaches. Her temperature at that time was not taken. Feeling that there was probably some obscure condition present, and unrecognized by me, I suggested that the patient call Dr. F. G. Hodgson, an internist. This she did, and an examination by Dr. Hodgson revealed nothing definite. He thought possibly it was a toxic headache from some faulty elimination. Coal tar products were given without any relief.

April 5th. A hypodermic of morphin was given for the first time, and this brought but slight relief. A blood examination was made which showed leucocytes 8000. No plasmodium malariæ.

April 7th. Patient put to bed, complaining of severe headache. Morning temperature, 101°; pulse, 100; evening, 101°; pulse, 98. Some sweating during the day. Bowels normal in movement, and nourishment taken at intervals. Aspirin five grains every three hours.

April 8th, patient complaining of headache and feeling

chilly. Examination of the ears showed both canals in almost
normal condition, as also the drum membrane. Blood count
showed leucocytes 10,000. Morning temperature, 101°; pulse,
98; evening temperature, 104¼°; pulse, 148. At one o'clock
this day the patient had the only distinct chill that occurred
during her illness. This was followed by a rise in tempera-
ture and pulse. No sweating occurred, according to the
nurse's report. Nourishment was constantly taken, and with
the exception of the pain at the back of her head, the patient
seemed fairly comfortable.

April 9th, patient had a very restful night. Complains of
the pain and tenderness at the back of the neck. Morning
temperature, 101 2/5°; pulse, 96; evening temperature, 103°;
pulse, 108. Quinin and strychnia were given constantly.

April 10th. Patient has been quite nauseated. Rested
quietly, but did not sleep. Mind clear. No pain about the
ears, and both canals begin to assume a normal appearance.
Morning temperature, 99 4/5°; pulse, 99; evening tempera-
ture, 100 3/5°; pulse, 90. Quinin, ten grains, has been given
three times daily, also full nourishment, and morphin only
when necessary. Mustard blisters were applied to back of
neck.

April 11th. Patient still complaining of severe pain in back
of head. Pupils normal and reacting to light and accom-
modation. No fundus changes found on ophthalmoscopic
examination. Morning temperature, 100°; pulse, 80; even-
ing temperature, 102°; pulse, 102.

April 12th. Patient slept very well, and seems brighter.
During the day she suffered with two attacks of nose bleed.
Constant stimulation; alcohol sponging at intervals. Differ-
ential blood count indicates a condition of sepsis. Morning
temperature, 103 2/5°; pulse, 118; respiration, 26. Evening
temperature, 103 2/5°; pulse, 134; respiration, 26. Exam-
ination of the lungs showed beginning pneumonia at the right
lower lobe. Patient thoroughly rational, but muttering when
asleep.

April 13th. Patient's condition much the same. Right
auditory canal looks normal, and only slight swelling in the
left. No discharge. Both drums looked apparently normal.
Patient listless. Fifty million of mixed vaccines were inject-
ed on account of the septic condition. Morning temperature,

99 4/5°; pulse, 108; respiration, 30. Evening temperature,
102 2/5°; pulse, 126; respiration, 30. Constant stimulation.
Patient perspiring freely. Decided consolidation in both lower
lobes of the lungs. From this time until the next night, April
14th, the patient rapidly became worse, passing into a coma-
tose condition and then death.

It was with great difficulty that we finally obtained permis-
sion to perform an autopsy, which was made by Dr. Claud
Smith, pathologist to Grady Hospital. The following is his
report:

"The calvarium being removed, an inspection of the men-
inges showed some congestion and edema. The substance of
the brain and ventricles was apparently normal. The right
lateral sinus, from a point just back of the bend before it en-
ters the mastoid groove, and extending almost to the torcular
herophili, contained a grayish semiorganized mass. About
the middle of this semiorganized mass was a collection of
about two centimeters of liquid pus. Smears upon slides
were made from this pus for microscopic examination. The
left lateral sinus contained a dark clot covered with a small
deposit of grayish material, in an almost similar position.
There was no evidence of adhesion between the meninges of
the brain and the right lateral sinus as the point of pus for-
mation. The dura mater did not show any evidence of in-
flammatory change. An opening was chiseled into the middle
ear and the mastoid cells, but no macroscopic evidence was
found of inflammatory condition or pus. The microscopic
examination of the smears of pus taken from the pocket in
the right lateral sinus showed an abundance of polymorphonu-
clear cells, many of which reacted only faintly to stains and
showed evidence of disintegration. Mingled with the pus cells
was an abundance of Gram positive staphylococci, some few
of which were intracellular. Smears taken were negative."

I take the liberty of making some additional remarks to
this report, as the writer assisted Dr. Smith in the autopsy.
The middle ears and mastoid cells were opened very cau-
tiously and thoroughly, and each structure was examined by
me with a probe. Absolutely no inflammatory signs could be
found showing any previous involvement of these parts. The
clots were limited to the lateral sinus, above and posterior to
the point where it turns down to form that portion of the

sinus which lies behind the body of the mastoid.   This latter portion showed nothing pathologic.

This case is unique from the fact that the infection and sepsis had its origin from a furunculosis of the auditory canal. That there was a bilateral infection is presumably evident from the autopsy, in that the thrombosis of the lateral sinuses occurred at almost identical points on both sides, and the fact that the clinical auditory canal symptoms were practically the same.   The question very naturally arises as to whether the lateral sinus thrombosis was a direct infection from the auditory canals, or whether there was first a general sepsis, and the thrombosis secondary or the result of this condition.

I think there can be no doubt as to the pneumonia being a septic one, the result of the general sepsis, and, on the other hand, I am equally convinced that the lateral sinus thrombosis was a direct infection from the auditory canals, and that it was from this point the septic material from the pus infiltrated the cellular tissue of the auditory canal, passed through the emissary veins, which perforate the bone at the mastooccipital suture and empties direct into the lateral sinus.   Such cases as this one are exceedingly rare, and the literature which I have examined mentions only one case which bears any resemblance to the one here reported.   This is found in Toynbee's old work on "Diseases of the Ear," published in 1860. Allow me to digress for a moment to say that this old work of Toynbee's could be well adopted today as a text book on the ear, for it contains the records of anatomic, pathologic and clinical work, which is simply marvelous to read, and it puts to shame most of the modern text books of today, which contain but little, if any, original investigations.   In this work I found the only mention of the subject in question.

Toynbee's discussion of this subject and the case reported by him throw so much light upon my own, that I shall quote very fully from his writings.   None of the modern text books have even mentioned the possibility of intracranial complications from diseased conditions of the auditory meatus, nor have they discussed the anatomic relationship between those parts.   On the other hand. Toynbee discusses the subject very fully.   He says: "The blood vessels ramifying through the membranous meatus are directly continuous with those entering and supplying the osseous meatus; the intimate connec-

tion between the dermis of the meatus and the bone is there-
fore very obvious. The relation of the osseous walls of the
external meatus to the cavity of the cranium are deserving
attention. In the adult it will be found that the upper wall of
the meatus consists of a solid lamina of bone, varying from a
line to two lines in thickness, which separates the cavity of
the meatus from that occupied by the middle lobe of the
cerebrum. In some cases a prolongation of the tympanic
cavity is found extending into the substance of the upper wall
of the meatus. In the child these relations differ remarkably
from those just detailed. At birth, and for the first year sub-
sequently, the only rudiment of the osseous external meatus
is the superficial depression situated in the middle of the outer
and lower part of the pars squamosa, immediately posterior to
the root of the zygomatic process. This depression, to which
the name fossa auditoria may be appropriately applied, has
the rudiments of the mastoid process posterior to it; its sur-
face is smoother, and its substance denser, it also contains
fewer foramina for the transmission of blood vessels, than
the surrounding bone. At the period of birth the portion of
bone forming the fossa is not more than half or three-
quarters of a line thick, the membranous meatus being at-
tached to the outer, and the dura mater of the middle cerebral
cavity to the inner surface. Its structure is far from being
compact or dense, and in its substance the blood vessels from
the meatus communicate with those of the dura mater. As
the bone approaches maturity, the fossa assumes an oblique
position and forms the upper wall of the external auditory
meatus, while it is separated from the cavity of the middle
cerebral fossa by a dense layer of bone, into which the cells
communicating with the tympanic cavity are not infrequently
prolonged. In the adult, the fossa auditoria has nearly lost
its oblique direction and become a horizontal lamina of bone.
From the foregoing remarks it will be evident that disease
of the membranous meatus externus is liable to extend to the
outer surface of the bone, and thence to the interior."

After making these anatomic observations, Toynbee reports
a case where the clinical symptoms and history undoubtedly
point to the diagnosis of sinus thrombosis, and where the
anatomic dissection showed that "the chief disease was found
in the external meatus, of which the membrane lining the

inner third was soft, highly vascular, easily detached from the bone, and covered by purulent matter. There was no appearance of ulceration on the surface." This is the only recorded case found in the literature which even resembles the history of the case here presented.

Kiechner,[1] in a very extensive article on diseases of the external ear, makes no mention of even the possibility of such a complication. All otologists have seen cases of severe furunculosis of the auditory canal which were difficult to diagnose from a mastoid involvement, especially when there was a history of a previous middle ear suppuration. In fact, a few cases have been reported where the outer table of the mastoid became involved from an extension of a suppurative process in the auditory canal, and where an operation was necessary.

G. Krebs,[2] in a very exhaustive article on "Rare Complications in Otitis Externa," mentions a case where the outer table of the mastoid became involved from this source, necessitating a mastoid operation. He also mentions the fact that such cases are extremely rare.

Von Leutert and Lannois have called attention to the fact that pus from a furunculosis of the external ear sometimes breaks through the wall of the external canal, and the abscess takes place behind the ear on the mastoid or in the fossa retromaxillaris.

Just a year ago I operated upon a child 8 years of age, where there was a typical subperiosteal abscess over the mastoid, with a history of a discharging ear. The auditory canal was swollen, thought to be secondary to the middle ear suppuration. The usual operation on the mastoid was started, when it was found that the pus came from between the membranous canal and the bone. The mastoid antrum and cells showed no involvement. It is by no means always easy to make a correct diagnosis in these cases.

Gruber has very justly called attention to the fact that we may have an intracranial complication where the middle ear has been previously affected, but at the time looks perfectly normal, and the external ear may show infection as the chief point of origin. In other words, the auditory canal infection is secondary to a previous middle ear suppuration which has entirely healed. That such is possible, we must admit, after

such a statement by so excellent an authority as Gruber; but it seems impossible for a thorough autopsy not to show even macroscopically some changes in the middle ear and the mucosa in the adjoining cells.

This case leads me to remark that the future text books must mention more in detail the possible complications of a furunculosis of the external auditory canal, and otologists must consider this process more serious than has heretofore been the case.

LITERATURE.

1. Kiechner: Schwartze's Handb. der Ohrenheilk.
2. Krebs, G.: Deutsche Therapeut. Monatshefte.

LXXIII.

FACIAL PARALYSIS IN MASTOID SURGERY.*

By CHARLES W. PERKINS, M. D.,

NEW YORK.

The consideration of facial paralysis in mastoid surgery with special reference to its avoidance as a sequel to operation is a subject lying so near the heart of every otologist that I need offer no apology for bringing the subject to your notice tonight.

Facial palsy appearing immediately after operation is generally attributed to nerve injury. That the surgeon may be unjustly blamed or unjustly blame himself is shown very plainly in the following case:

An adult, male, was operated upon for simple mastoid by a surgeon of great skill. Immediately after the operation a complete facial paralysis was noticed on the involved side. As the operator was sure that he had not injured the nerve in the mastoid or middle ear, he concluded that the injury must have been in the neck, although proper care had been taken while freeing the tip with scissors. There was an epidural and perisinous abscess of considerable size. The paralysis persisted for about six weeks, and then during the next three weeks totally disappeared, proving that the nerve was not injured in the neck, but in all probability the epidural abscess in the posterior fossa had extended to the internal auditory meatus and the facial had become involved at this point.

In a similar manner in a case which has come under my observation, the nerve has become involved at the geniculate ganglion. The patient had a large extradural abscess in the middle fossa, and facial paralysis which cleared up soon after operation.

Nevertheless, it is a rule to which exceptions are rare, that

---

facial paralysis showing itself immediately after operation, i. e., when the patient emerges from the anesthetic, offers a bad prognosis, indicating some injury to the nerve, while those appearing later, being due in all probability to an inflammation of the nerve or its surroundings, generally clear up in time.

The avoidance of facial nerve injury and consequent paralysis in the simple mastoid operation, would seem to be an easy matter. Yet one sees from time to time cases in which the facial nerve has been injured in this procedure. In infants in making the initial incision one must bear in mind the fact that the nerve lies more superficial than in the adult, and unless due care is taken it may be injured at the lower end of the incision. Before the mastoid tip is formed the nerve does not become deeply placed, and in order to avoid its injury the lower end of the incision should be made posterior to its usual position in adult operations.

That this danger is a real one was demonstrated to me in a case coming under my observation where the operator in extending the lower end of his incision in an infant took a direction somewhat forward and the nerve was severed, with consequent permanent facial paralysis. Then, again, the nerve may be injured beneath the horizontal semicircular canal. Perhaps I may say, only by one unfamiliar with the mastoid operation. I have myself recently done a secondary operation on a child, two years old, which had been previously operated upon in one of our large hospitals by a general surgeon. In this case the facial paralysis was noticed immediately after the primary operation. The antrum was opened, but the posterior wall was taken down in such a manner that I feel sure that the nerve was injured immediately beneath the semicircular canal. A considerable number of involved cells were found which had not been molested at the previous operation. I am satisfied that the desire to open the antrum as the object of the mastoid operation (an evident misconception) is responsible for facial nerve injury in a certain number of cases.

In cellular mastoids, especially in mastoiditis of the Bezold type, one frequently finds himself working very near the facial nerve, sometimes only a thin layer of bone intervening between the wound and the nerve. In such cases I have contented myself with leaving the thin layer of bone and have not had cause to regret it. While the object of the mastoid operation is to

thoroughly ablate the cells and clear out all infected areas, one cannot follow up every red spot, no matter where it may lead. There are cases in which one's industrious perseverance may become reprehensible, and when it results in injury to the facial nerve it is one of these.   One's knowledge of anatomy should teach him when to stop, and if it does not he may have the mortification of a postoperative facial paralysis.

In "freeing the tip" with the scissors, one may cut the facial nerve if he does not hug the bone closely.   I have been in the habit of separating the muscles from the mastoid tip with the elevator.   I have found this elevator (figure I) very useful for this purpose.   With it one can do all of the work to be done with any elevator, besides it is sharpened so that it may be used in either direction.   Of course, in the radical operation it is necessary to use a smaller elevator of different type to separate the canal wall.   Here an ordinary elevator used in the submucous resection of the nasal septum will answer very well.

When we come to do the radical operation we are working much nearer to the facial nerve.   Someone has said that if we injure the dura our patient may die, if we injure the sinus our patient may die, but if we wound the facial nerve we will wish that he had.   This perhaps too forcibly expresses one's chagrin at the occurrence of this accident, yet a postoperative facial paralysis that one feels responsible for is a very disquieting occurrence.

We must remember that the facial nerve passes horizontally, or nearly so across the internal wall of the tympanic cavity between the oval window below and the horizontal semicircular canal above.   That in this part of its course it is covered by a thin layer of bone easily fractured, and not infrequently this layer is absent or presents dehiscences.   It turns downwards immediately beneath the horizontal semicircular canal, taking a course internal to a line extending from the prominence of the canal and parallel with the median line.   The only steps of the radical operation in which it can with any probability be injured are: First: Taking down the bridge.   Second: Lowering the facial spur.   Third: Curetting the tympanum; and Fourth: Curetting the eustachian tube.

In all descriptions of the radical mastoid operation with which I am familiar, the directions given, after opening the antrum, are to take out a wedge-shaped piece of bone in which

the apex of the wedge should be at the aditus ad antrum. I am
satisfied that in carrying out this procedure in this manner there
is great danger of injuring the facial nerve at the apex of the
wedge, either if it is pointed too low, or if the aditus should be
very small. To obviate all danger of this accident I would ad-
vise the following procedure: After opening the antrum the
opening is enlarged well over towards the zygoma until the
tympanic vault is well exposed, leaving at the same time the
superior wall of the canal or at least the internal part of it.
(This step is well shown in figure II.)

Now we have constructed a bridge of which the posterior
abutment lies at the aditus ad antrum, and the anterior at the
anterior tympanic wall. It is evident that the facial nerve lies
immediately beneath the posterior abutment of this bridge.
Now we work forward and take down the bridge as far an-
terior to the posterior abutment as possible. This may be done
with the chisel, curette or rongeur forceps, my preference be-
ing for the two former. When this bridge is broken through
(as shown in figure III) the overhang represents safety to the
facial nerve. If this procedure is carried out as above de-
scribed, the only danger to the facial nerve would come from
one's chisel slipping, or the back of one's curette pressing upon
the nerve as it crosses the tympanic cavity, both of which acci-
dents can be avoided with proper care.

Next we lower the facial spur that proper healing may take
place. For this purpose a chisel may be used, being careful
that it is well under control, or what the author prefers, a
Richard's curette of proper size. If one simply removes the
overhang and is careful that all of the force is exerted out-
wards and with the fulcrum on the upper border of the bone
wound, that is, the squama, he need have no fear of injuring
the nerve. It is only when the curette rotates so that the force
is exerted internally, the internal tympanic wall being allowed
to become the fulcrum, that the nerve is menaced. The pos-
terior canal wall may be lowered as much as desired, only bear-
ing in mind, for safety sake, the rule above given, i. e., that the
facial nerve lies internal to a line dropped from the horizontal
semicircular canal parallel with the median line of the body.

In curetting the tympanum one should use a ring curette
with flexible shaft, and should even then watch for twitches of
the face, as there may be a dehiscence in the fallopian canal.

I have known a facial nerve to be irreparably injured by a single stroke of a stiff curette, the nerve being severed in its middle course.

Then in curetting the eustachian tube it should be remembered that the back of the instrument can press upon the fallopian canal in the middle ear. I have known this to occur in a case operated upon by a man of great experience. The paralysis was fortunately temporary, passing away in about six weeks. In this case there was produced, no doubt, a depressed fracture of the bone overlying the nerve.

In the labyrinth operation of course one works very near the facial nerve, and in some cases perhaps it is impossible to avoid injuring it. Still one should do all in his power to save the fallopian canal, and I believe it can be done in almost every case. The nerve will do much better lying in its natural surroundings so far as they can be left; and sufficient drainage of the labyrinth can be obtained with the bridge containing the fallopian canal left intact.

The author has performed fifty-five radical operations without mortality or facial nerve paralysis attributable to the operation. In two of my cases facial paralysis has developed after the operation. The first was a girl of eighteen. After I had removed the bridge the assistant packed a gauzs sponge into the tympanic cavity. This simple procedure produced a well marked twitch of the facial muscles. Having had this warning, I was very careful of my intratympanic manipulations. Nevertheless, on the following day, some twenty hours after the operation, there was a partial facial paralysis, which became complete about two days later, indicating without doubt a neuritis of the facial nerve. The palsy passed away in about six weeks. There was doubtless in this case a dehiscence in the fallopian canal, and had I used a stiff curette and some force in cleaning the middle ear, I would have done irreparable injury to the nerve. The second case, a boy of about thirteen years, developed a facial palsy about six weeks after operation. This cleared up perfectly in about five weeks.

Included in the above fifty-five cases are two complete labyrinth operations, in which the canals were ablated and the capsule of bone overlying the first two cochlear turns was removed.

In doing these operations on the temporal bone I have found indispensable a set of gouges which I have devised that are

made for me by Tiemann & Company (figure IV). The distinctive feature of these are the fact that they are beveled in front. This enables one to take a very thin shaving of bone, and the force is exerted upon the chip removed and not upon the bone left behind, enabling one to work much nearer to important structures without danger and reducing traumatism to a minimum.

In conclusion, I would say that I am led to believe that facial injury can be avoided almost entirely in operations on the temporal bone, and the directions above will be found useful for one trying to reduce the number of these injuries to the minimum.

FIGURE I.
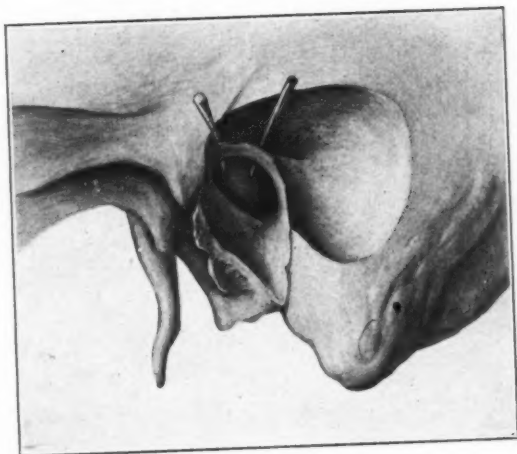
FIGURE II.
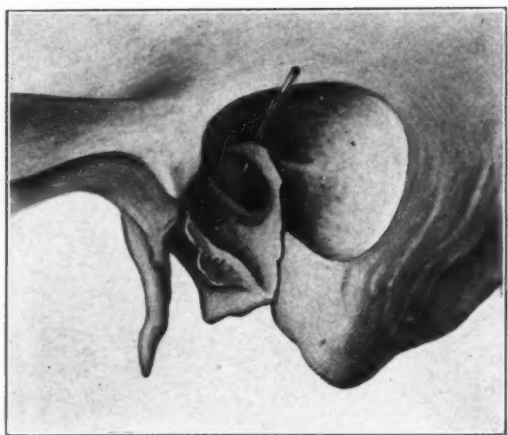
FIGURE III.
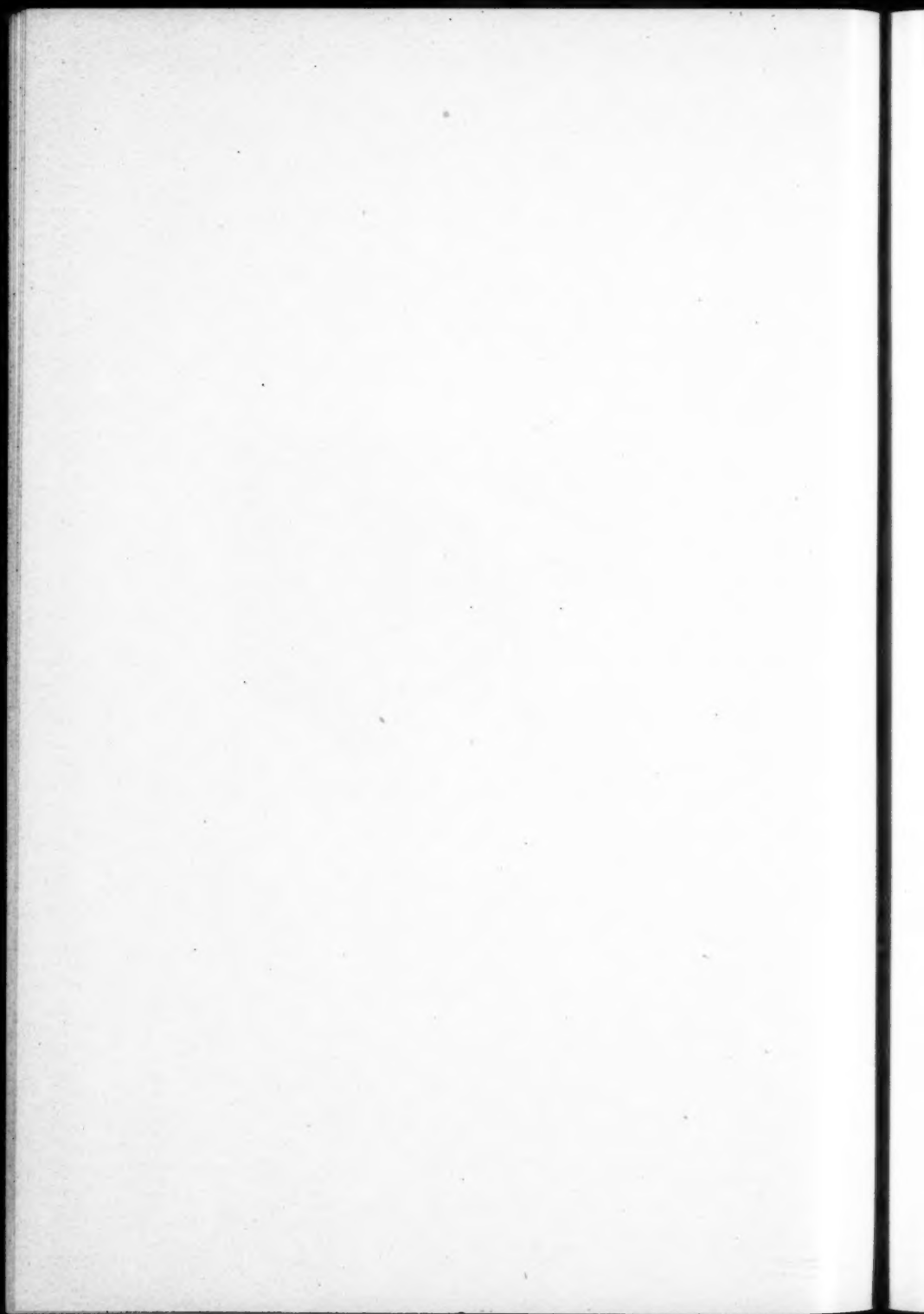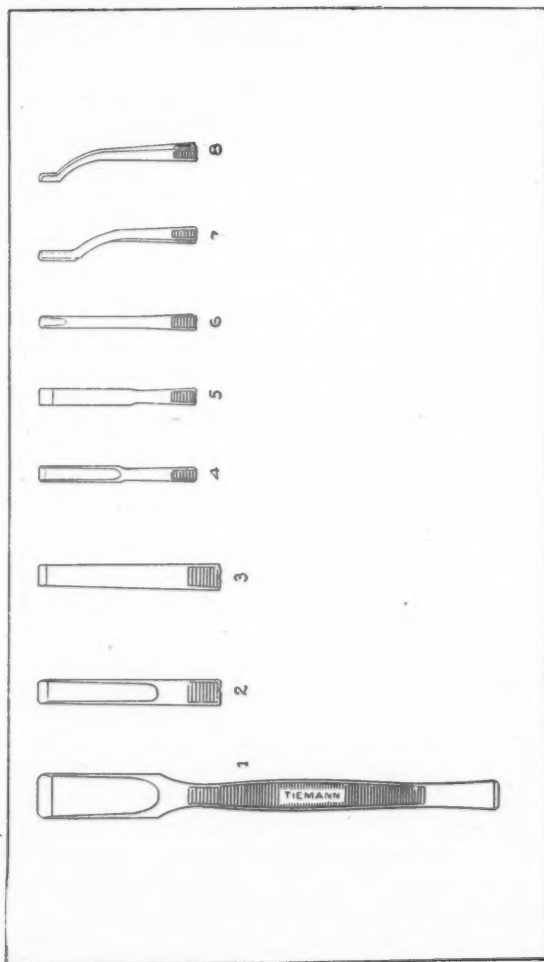
FIGURE IV.
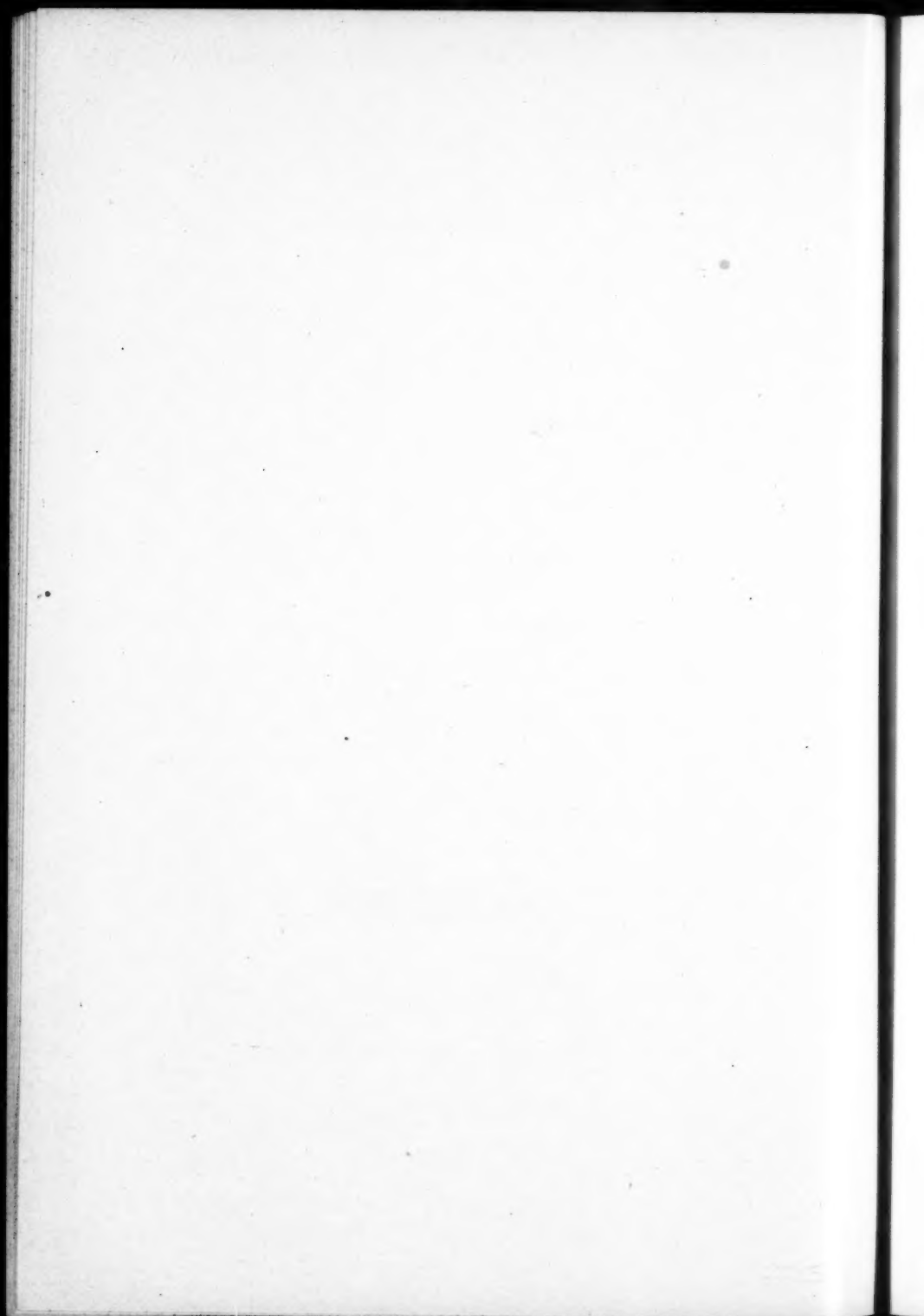
THE CUBICAL CAPACITY AND SUPERFICIAL AREA OF THE SPHENOID, MAXILLARY AND FRONTAL SINUSES.*

By Hanau W. Loeb, M. D.,

St. Louis.

This work followed a study of the anatomy of the accessory sinuses of the nose from the standpoint of their greatest diameters, anteroposterior, superoinferior, and lateral. It is based on a method which was described before the Third International Congress at Berlin in 1911.

At that meeting I showed how a complete plaster cast of a sinus could be made by uniting plaster moulds of those portions of a given sinus lying adjacent to one another in serial sections of the head.

These portions can be readily removed without injury to the specimen, affording a great advantage over the old method of making a cast with Wood's metal, which necessitates the destruction of the specimen before the removal of the cast. The cubical capacity is easily ascertained by the old method of determining the amount of water displaced by the cast.

The superficial area presents a much more difficult problem. After many experiments a plan was finally devised which was considered sufficiently accurate to justify its adoption.

The cast is covered with strips taken from a known quantity of adhesive plaster, fitted accurately without stretching. Subtracting the amount remaining from the known quantity gives the superficial area of the sinus in question.

The results of the study of the sphenoid sinus (except four which are added) with the methods were presented by the writer at the Congress; those of the maxillary are from studies by Dr. Virgil Loeb; those of the frontal by Dr. Albert Miller.

SPHENOID SINUS.

The variation in form is exceedingly marked, in fact, it may be said that there is no typical shape.

In all the specimens the inner wall is almost uniformly smooth and free from prolongations, while the other walls may be exceedingly irregular. They exhibit projections externally, anteriorly, posteriorly, and even inferiorly.

I have already called attention to the extreme variations, both in size and shape, in the sphenoid sinuses of various heads, as well as in individual pairs. Why there should be this great difference at the very center of the skull is a problem still to be solved.

The cubical capacity of the sphenoid sinuses examined is as follows:

Head.	Right. cc.	Left. cc.
III	3.5	1.0
VII	6.5	9.9
VIII	3.8	3.0
IX	2.4	2.3
XI	4.0	4.5
XII	0.6	7.5
XIV	6.3	1.0
XXI	8.0	2.9
XXIII	3.0	3.1
XXVI	11.8	1.9
XXVII	4.8	8.2
XXXV	8.3	10.0
Average	5.25	4.61

Average of all the sinuses, 4.93. Extremes: Head XII, right 0.6; and head XXVI, right 11.8.

These differ but little from the average of the ten heads previously presented, and still show that however much the sphenoids differ from one another in size and form, the average of those of each side remains about the same, approximately 5 cc.

The superficial area of the sinuses is as follows:

Head.	Right. sq. cm.	Left. sq. cm.
III	16.0	5.6
VII	26.7	27.2
VIII	13.9	12.9
IX	8.9	9.7
XI	13.2	16.5
XII	2.4	22.7
XIV	24.7	4.0
XXI	18.3	10.8
XXIII	10.5	12.2
XXVI	26.4	7.4
XXVII	17.3	21.8
XXXV	25.5	28.2
Average	16.99	14.92

Average for all the sinuses, 15.92.

According to the observation thus far made, the superficial area of the right sphenoid sinus is a trifle greater, on the average, than that of the left side.   It must be admitted, however, that there is a great difference in the two extremes; head XII, right 2.4; and head XXXV, left 28.2.

MAXILLARY SINUS.

The maxillary sinuses, as shown by the casts, are roughly triangular in shape, with the apex posterior, with all the walls practically triangular, except the internal, which is more or less quadrilateral.

The following indicates the cubical capacity of the forty-two examined:

Head.	Right. cc.	Left. cc.
III	8.0	7.5
IV	24.8	24.5
V	24.5	19.2
VI	16.8	13.0
VII	13.9	14.5
VIII	7.0	4.9
X	10.2	10.0

Head.	Right. cc.	Left. cc.
XI	14.5	15.2
XII	10.0	7.2
XIV	13.0	14.8
XV	13.0	9.6
XVI	4.5	4.8
XVII	12.0	8.0
XVIII	11.5	4.8
XIX	12.5	18.5
XX	14.9	15.2
XXII	14.7	8.5
XXVIII	11.0	9.0
XXIX	8.8	7.5
XXX	18.3	15.0
XXXI	9.5	6.2
Average	13.02	12.85

General average, 12.94.

While the extremes are head XVI, right 4.5, and head IV, right 24.8, the average for the right is very near that of the left.

The superficial area of the sinuses is as follows:

Head.	Right. sq. cm.	Left. sq. cm.
III	25.9	25.2
IV	52.3	48.4
V	51.3	45.8
VI	43.1	37.0
VII	34.2	37.7
VIII	27.7	21.2
X	29.0	29.7
XI	35.1	39.9
XII	28.3	22.4
XIV	35.2	35.0
XV	33.1	28.6
XVI	12.1	16.3
XVII	30.0	25.0
XVIII	34.2	16.5
XIX	31.0	40.0

Head.	Right. sq. cm.	Left. sq. cm.
XX	33.1	33.1
XXII	36.9	27.3
XXVIII	29.1	26.3
XXIX	25.9	25.9
XXX	41.4	38.1
XXXI	27.4	21.0
Average	32.91	30.43

General average of all the sinuses is 31.68, showing a marked uniformity in the two sides.

The extremes are head XVI, right 12.1, and head IV, right 52.3.

FRONTAL SINUS.

The casts of the frontal sinus show that Hajek's division of the varieties into first, second and third degree is justified. The simplest form is that of a prism flattened antero-posteriorly, rounded superiorly and greatly narrowed inferiorly. In the larger and more complicated varieties there is merely an addition of one or more similar sections placed external to the primary.

The cubical capacity is as follows:

Head.	Right. cc.	Left. cc.
V	8.2	5.0
VII	3.8	3.6
X	1.9	2.2
XII	1.9	3.4
XIV	7.0	4.9
XVII	1.8	4.2
XVIII	4.0	3.2
XXVIII	2.4	3.7
XXIX	2.3	3.3
XXXI	0.9	3.9
Average	3.42	3.74

General average, 3.58.

The extremes are head XXXI, right 0.9, and head V, right 8.2.

The superficial area is as follows:

Head.	Right. sq. cm.	Left. sq. cm.
V	32.3	26.6
VII	16.3	17.6
X	9.8	11.5
XII	7.9	16.7
XIV	28.6	21.1
XVII	8.5	17.5
XVIII	14.5	12.9
XXVIII	10.6	15.1
XXIX	11.0	14.2
XXXI	5.5	8.5
Average	14.50	16.17

General average, 15.34.

The extremes are head XXXI, right 5.5, and head V, right 32.3.

It will be observed that in selecting the sinuses for measurement there has been no attempt made to group them so as to determine the total cubical capacity and superficial area of the sinuses on the right and left sides. However, what has already been done warrants the hope that some interesting facts are to be brought out by such investigation.

LXXIV.

CASE OF CONGENITAL BILATERAL MICROTIA WITH TOTAL OSSEOUS ATRESIA OF BOTH EXTERNAL AUDITORY CANALS.*

By John Randolph Page, M. D.,

New York.

This patient is now three years and nine months of age. There is no history of deafmutism or deformity of any kind in either branch of his family. His father was fifty-three years of age when he married, and his mother thirty-three, making a difference in their ages of twenty years. Ten days after the marriage the father complained of headache in the evening, and died of cerebral hemorrhage that night. I first saw the child when he was seven weeks old, and watched him off and on for two years, and at the end of that time I was confident that he had some hearing. Dr. Cole, who made an X-ray picture of his head, thought he could demonstrate a tympanum and ossicles.

In order that all possible aid to hearing might be given to enable the child to talk and develop mentally, it was deemed wise to operate on one ear and, if improvement was noticed, later on to operate on the other.

An incision was made over the mastoid and the rudimentary auricle was displaced forward; the bony meatus was found to be entirely closed and its location was only evidenced by a small dimple in the bone itself. No evidence of a fibrous canal was present. The cortex of the mastoid was removed and every cell beneath it was found filled with a thick mucopurulent fluid. A fairly large antrum, filled with the same material, was found, and from this a small aditus leading to a contracted middle ear.

It is considered good surgery, I believe, to end the operation

*Presented before the Section on Otology, New York Academy of Medicine, October 12, 1912.

at this point and not invade the middle ear, as it is thought best to allow sound waves to reach the internal wall of the tympanum through the aditus by way of the autrum which alone is opened. Before operating on this case, however, I assisted Dr. Duel in a similar one in which he followed the method referred to, and experience with it convinced me of the difficulty of keeping the aditus open even with daily dressings and the exercise of the greatest care. I therefore decided in this case to make, if possible, a large radical cavity and skin graft.

The bone corresponding to the location of the bony meatus was cut down to the aditus, making a large single cavity in the bone. No evidence of fibrous canal lining, or drum membrane, or annulus was found. In removing the bone from along the line of what should have been a bony canal down to the aditus to the region of the external semicircular canal, which was visible, a hard tubular line of bone was encountered external to the horizontal semicircular canal, and it was at first thought to be the fallopian canal, despite its faulty position. More bone was removed, however, and it was found to be the edge of a rudimentary incus which was imbedded in the bone. No evidence of a membrane or of the other two ossicles was discovered. The middle ear cavity was a narrow, slit-like cavity from without inward, but fairly wide from the orifice of the eustachian tube back to the aditus and from above downward. The eustachian tube was patent and the tympanum was lined with membrane continuous with that of the tube, and the whole cavity was filled with the same mucopurulent fluid that was in the antrum and cortical cells.

A large meatus not less than one-half inch in diameter was cut in the rudimentary auricle, the skin being turned in as far as possible in order to retain a new meatus, as there was no cartilage to support it. The posterior incision was closed and the cavity was skin grafted ten days later. A caloric test after the operation was markedly positive.

It has now been nearly two years since the operation was performed and the meatus easily admits a medium sized ear speculum and is, without stretching, about three-sixteenths of an inch in diameter. The interior of the cavity is clean, white and dry; a medium sized radical cavity with a closed tube. His hearing, his mother says, is decidedly improved, and his appre-

ciation of words and sounds increases from day to day. During the past six months he has begun to form short sentences. Before he was operated on he said nothing except "mamma." In May, 1911, the baby returned to his mother's home, and after being there a month his vocabulary began to develop. He began to repeat words spoken to him by his mother. A year ago he was able to call his brother Joe by name. During the past six months his improvement has been more marked and he forms a few short sentences of simple words. His mother states that his attention is called to her voice more quickly than to that of other people (naturally). When his attention is attracted to her, she finds it unnecessary to raise her voice. He is attracted quickly by a moderately low whistle.

I should like to know the opinion of the Section as to the advisability of operating on the other ear. The mother wishes to have it done, as she is convinced that his hearing was improved by the first operation.

LXXV.

SINUS THROMBOSIS—GRUNERT'S OPERATION—RECOVERY.*

By Frank Tucker Hopkins, M. D.,

New York.

Grunert's operation, socalled from Prof. Grunert of Halle, a/S., Germany, who in 1901 suggested it,[1] is the free exposure of the sinus and jugular bulb by the removal of the outer bony wall of this venous channel, together with the outer wall of the jugular foramen, thereby making an open gutter of bone in which lie the sinus and jugular bulb; and hence the operation is also called the "gutter operation."

The operation is generally done from without inwards, working through the overlying integment and muscles down to the bone and then removing the bone from below upwards. Voss, however, in 1904[2] suggested that the operation be done from the sinus end, working downwards and not dividing the overlying soft parts. He considered it an easier method and one resulting in less deformity.

The chief dangers of the operation lie in the possible wounding of the facial nerve and in a hemorrhage from the inferior petrosal sinus, if the jugular clot has not extended into this sinus sufficiently far to shut it off.

Although a well indicated operation, it has not, however, often succeeded in saving the patient's life. I am inclined to think that this is because it is generally undertaken too late, when general septic infection has already begun.

In February last I was called to see a seven-year-old child who was showing all the symptoms of acute mastoiditis on the left side. Eight weeks before he had developed scarlet fever, and from that had come an acute middle ear suppuration, now three weeks old. The discharge was abundant, the upper and

---

posterior walls of the canal were bulging, and there was marked tenderness over all the mastoid, with a slight beginning redness of the mastoid integument. The patient's (rectal) temperature was 102.4° F.; pulse, 120; respirations, 26. There had been severe pain in the ear since the previous day.

The patient was at once sent to the New York Eye and Ear Infirmary and I operated that same afternoon. Examination of the pus from the mastoid wound showed a streptococcus infection; the blood count was 9,400 white, with a differential of 68 per cent polynuclear cells. The mastoid was much infiltrated with pus, while a large perisinus abscess had already developed. The sinus was not healthy in color, rather gray, but evidently contained fluid blood, and so no exploration of it was made.

During the next four days the temperature fell steadily and the patient's condition was satisfactory. On the fifty day, however, the temperature suddenly rose to 106.2°, and during the next twenty-four hours showed the usual severe fluctuations which indicate sinus thrombosis. The blood count was altered, the white numbering 14,800 and the polynuclear rising to 91 per cent. A jugular operation was done. In this operation the vein was found to be quite empty and was resected. The sinus was curetted well down into the bulb without bleeding, and back for an inch towards the torcular until free bleeding occurred. Examination of the vein and of the clot from the sinus showed no infection. But the temperature after this operation remained down only one day and then rose to 105.2°, falling soon after to 98.2°. Culture of the discharge from the open sinus showed streptococcus infection towards the bulb and towards the torcular.

Acting on the advice of Dr. Dench, who kindly saw the case with me, I made a surgical exposure of the jugular bulb, or, in other words, I did the Grunert operation, proceeding however by the method suggested by Voss, i. e., from above downward. Just at the conclusion of the operation a very severe hemorrhage from the inferior petrosal sinus weakened the patient's pulse so seriously that a free operation towards the torcular, which I had expected to do, had to be postponed. However, enough of this clot was removed to obtain a little bleeding.

No facial injury resulted from the "gutter operation," and the patient slowly recovered from the hemorrhage. Still, al-

though he improved in strength, yet every day there was an elevation of temperature, which, though slight at first, later became 104° and 105°. The blood count, however, during the next day was good, the white cells numbering 10,500 and the polynuclear being 65 per cent. Moreover, there was no longer any streptococcus infection down in the bulb. I felt, therefore, that the trouble lay in the posterior clot extending towards the torcular, and so decided to make a freer exposure of this. Accordingly the boy was again etherized and the sinus exposed and curetted for full two inches further back, until the bleeding was very free. The patient made a good recovery from the operation and there was no further elevation of temperature or occasion for further anxiety. Some two weeks later I did a plastic operation on the open and granulating surface, which added much to the comfort of the patient and materially shortened the time of healing.

I desire to thank Dr. Dench for his interest and advice in this difficult case, and also to express my obligations to Dr. Perkins, who assisted me at these operations.

REFERENCES.

1. Grunert, K.: Original article, Archiv f. Ohrenheilk., vol. 53, 1901, p. 286.
2. Voss: Zeitschrift f. Ohrenheilk., vol. 48, 1904. p. 265.

LXXVI.

A CASE OF EARLY OTOSCLEROSIS TREATED WITH RADIUM.*

By Richard Lake, F. R. C. S.,

London.

The patient, a young lady of nineteen, of particularly good physique, was first seen by me on May 24, 1909, on account of increasing deafness of the right ear of two years' duration, and of a slight recent depreciation of her auditory power on the left side, which she had noticed for about two months. The history showed a strongly marked inherited tendency on the part of the patient to abnormal influences in her nervous organization. For example, at one time she had become very thin, for no particular reason, and had remained so for a long time. At other times she was very much disturbed by loud noises; these seemed to induce something approaching a nervous shock, especially when sudden and unexpected. But in all other respects she was a remarkably capable person, and one would naturally remark on her intelligence and capability.

The first examination gave the following results:

2″	Acoumeter (Politzer's)		3′
4′	Voice	Room	16′
0	Whisper		5′
Neg.	Rinne C. (128)		Pos.
Neg.	Rinne C^2. (512)		
18½″	Bone Conduction		16″
27000	Galton (Edelmann)		41000

AIR CONDUCTION.

$$
\text{Abs.} \left\{ \begin{array}{l} \text{C. 16. d. v.} \\ \text{C. 32. d. v.} \\ \text{C. 64 (16″)} \\ \text{C. 128. c.} \end{array} \right\} \text{Abs.}
$$

$$
\begin{array}{c}\text{Heard}\\\text{but diminished.}\end{array} \left\{ \begin{array}{l} \text{C. 256.} \\ \text{C. 512.} \\ \text{C. 1024.} \\ \text{C. 2048.} \end{array} \right\} \begin{array}{c}\text{Heard}\\\text{but diminished.}\end{array}
$$

---

*Read before the Ninth International Otological Congress, at Boston, August 12-17, 1912.

The objective examination showed extremely translucent tympanic membranes with freely mobile mallei. On the promontory of the right side there was an unusually pronounced red blush, whilst there was at the same time a blush on the left promonotory, though less in extent and intensity. The eustachian tubes were perfectly free. The diagnosis was of commencing otosclerosis on the left side, and of rather more advanced otosclerosis on the right side; and this despite the absence of paracusis Willisii and the fact that the bone conduction on the left hand side was just about 64 double vibrations, and although the bone conduction was very much diminished. This marked depreciation of the bone conduction was attributed to neurasthenia.

After that visit in May, 1909, I did not see the patient again until June 8, 1910, nearly fourteen months later; and the reason given for this long absence was that the patient's previous aural experience had been of so unpleasant a nature that she feared to undergo treatment, although she was aware that her hearing was steadily deteriorating. The treatment to which she had been subjected in the provinces had been that of simple Politzerization, but this had so distressed her, on account of the sudden noises accompanying the inflation, that she was quite unable to bear a probable repetition.

For some reason or another the tests I made on the patient's second visit were very briefly recorded; merely that the voice had fallen to 1½ feet on the right hand side—that is, ordinary conversation voice. On objective examination, however, one found a marked increase in the area of redness, which had been, as stated above, only in the region of the promontory. The left ear was now as far advanced in appearance as the right had been at the time of the first visit, while the redness had considerably extended on the right side. Treatment was now undertaken. Otomassage with injections of iodid of mercury, two or three injections a week, were employed for two weeks, together with reeducation by means of an Eolian organ. At the end of this treatment no improvement whatever was noticed, nor was the rate of increase of the red patches on the inner tympanic wall checked. Entire rest was then tried, as the patient was again losing flesh rapidly, and she was sent into the country, where she remained from the middle of August till the beginning of November. On

November 2, 1910, a further examination was made, and one found that the whole of each inner tympanic wall was equally red. There was no pain, no increase of temperature, no increase of pulse rate, or any other inconvenience whatever. Examination then showed on the right hand side that, to Rinne's test with a C tuning fork of 512 double vibrations, an indefinite result was given, instead of a definite negative. A whisper was heard at 3 feet on the left hand side; the patient was able to perceive C 128 double vibrations on the right hand side, and C 64 double vibrations on the left, as the respective lower limits of hearing. The result of these tests makes it even more annoying that one had no record of the tests made at the previous visit; but, at that time, I had no suspicion that the case was going to prove of such exceptional interest.

At this time it could be said that, despite both attempts at treatment and the results of rest, one had obtained no further improvement, except the question as to the indefinite result of Rinne's test in the left ear; this test was no longer definitely negative; whereas the previous red patches on the inner walls now covered the whole inner tympanic walls, a condition which I had certainly never seen before, and to my knowledge no such condition has been reported.

Having failed in my endeavors, I had now only one idea left as to how to deal with this condition; this was the use of radium. It was fully explained to the young lady's parents how speculative the nature of this treatment was, and how uncertain was the result. The parents, however, were as anxious as I was to get a good result, and were quite willing to take any reasonable risk. The idea underlying the application of radium was that the whole of this redness was due to a mucoperiostitis of the tympanum, and consequently the whole of the reddened portion was formed of inflammatory or granulation tissue of the deeper layers of the mucoperiosteum; and that this was probably accompanied by a certain amount of new bone formation, together with rarefying ostitis, which should be readily reduced by the use of the radium rays.

I obtained the assistance of Dr. Iredell, the X-ray expert at Guy's Hospital, London, and he undertook the treatment, at first alone. He used in either ear an equivalent amount of radium sulphate, that is to say, half a milligram. The

radium used in the left ear was enclosed in a glass tube, and in the right in a small silver tube. The tubes were about one-eighth inch in diameter, the glass tube being nearly one inch long, whereas the silver tube was not more than one-fourth inch in length. Later both of these tubes were covered with a piece of india rubber tubing. The duration of the treatments varied from ten to twenty minutes. Altogether there were seven treatments given between the 10th of November and the 9th of December, and, from the results, I considered that the tube had not been introduced sufficiently far into the canal. So, after December 9th the patient was treated in my consulting rooms, under my direction, for the length of time, judged by the effects of the previous treatments, adopted by Dr. Iredell. From that time forward the silver tube alone was used, and at this time we took the second record of the bone conduction; on both sides the bone conduction had fallen to 14 seconds on the right side and 12 on the left. Of this, however, I took but small notice, for the reasons alluded to before. The duration of this same tuning fork on the left side by air conduction was 24/60ths, or 24 seconds. Between the 9th of December and the 18th of February, 1911, nine further treatments were given, the same time duration being observed; and, at the end of that time, the patient was able to hear a C of 32 double vibrations on the right hand side.

The patient then had, for some reason or other, another of those peculiar attacks of less of flesh, so she was sent away for another rest until the 21st of March. On her return she had three more treatments to each ear, of fifteen minutes each. There was a particularly well marked reaction noticed on the 14th of April, when treatment was recommenced; and twenty-five minute treatments were given on seven occasions, ending on July 19th. At that time the bone conduction had risen on both sides to 20 3/5ths seconds, and the air conduction of the same fork on the left side to 36 seconds. By this time the ears had become quite normal in appearance, as, during the treatment, the tubes had been directed towards the part which showed the greatest tendency to resist the action of the rays. There was a gradual contraction of redness and its gradual limitation to the promontory, especially on the right side; the last place to become normal in appearance being

the edge of the round windows.   The final examination with
regard to the voice test showed that the patient was able to
hear it at three inches on the right hand side, and a whisper
at eight to fifteen inches on the left.

In my experience, these local red patches on the promon-
tory and in the vicinity of the round windows are distinctly
uncommon.   I do not remember ever having seen one in a
male, nor have I been able before this, in those cases which
I have seen, to get rid of these red patches, nor to check the
progress of the disease.   It certainly seems to me that, if
this is otosclerosis, it must be extremely rare, without there
are other cases in which this red reflex is never seen.   There
is also no doubt in my mind that if this treatment had been
adopted in this case when it was first seen in 1908, the neces-
sary use of radium would have been short, and the results
might have been very brilliant.   The patient has recently
returned from India, and I append a full investigation of her
powers of audition.

	P. W.	
	Weber	
4″ to 6‴	Voice	room (16 ft.)
	Whisper	20″
	Acoumeter	
n	Rinne (128)	p
p/n	Rinne (512)	
22″	Bone condn.	22″

AIR CONDUCTION.

	C	16	vibs.	abs.
Abs.	"	32	"	+
	"	64	"	+
	"	128	"	+
	"	256	"	+
Dim.	"	512	"	+
	"	1024	"	+
	"	2048	"	+
Abs.	"	4096	"	+
	"	8192	"	+

LXXVII.

A CASE OF THROMBUS AND REMOVAL OF INTERNAL JUGULAR VEIN.

By Frank Allport, M. D.,

Chicago.

Mrs. V. K., age about 35 years, was first seen by Dr. Tibbits of Menominee, Michigan, May 13th, 1912. She reported that she had "caught cold" and had a severe pain in her right ear. In about three days this was followed by a discharge. For about three weeks the pain was evident at irregular intervals and the discharge continued constantly. She then consulted Dr. Tibbits, who ascertained that she had a temperature of 103.8° and a pulse of 100.

She was sent to the hospital and Dr. W. O. McBride of Marinette, Wisconsin, was called in consultation. He discovered a small perforation in the anterior inferior quadrant of the drum head, which he materially enlarged. There was slight sagging of the posterior superior wall of the meatus and some tenderness over the mastoid tip, but no swelling of the mastoid tissues. That evening the temperature advanced to 105° and the pulse to 120. Before morning the temperature fell to 103°, but in a few hours advanced again to 105°.

On May 14th Dr. McBride performed a simple mastoid operation and found pus in the cells and a healthy appearance of the bone covering the sigmoid sinus. The temperature rapidly dropped to 101° and the pulse to 100. From this on the temperature and pulse made rapid upward and downward flights, and on May 17th, 19th and 20th she had chills. Her mental condition remained clear.

On May 20th I removed a large thrombus from the sinus and secured a free flow of blood from both the torcular and bulbar directions. Her temperature immediately dropped to 99.8° and her pulse to 100. Both temperature and pulse gradually became lowered to almost normal until May 27th, when she had another chill and her temperature shot up to 105° and

her pulse to 150.   By the next day the temperature was 100°
and the pulse 100.   May 29th she had another chill, followed
by another, and her temperature reached 107° and her pulse
160.   That night when I saw her she was in no material pain
and was clear mentally.   Her temperature was 100° and her
pulse 110.   The differential blood count, made by Dr. E. V.
McComb of Menominee, showed the polynuclear cells to have
a percentage of 99 per cent.   There was no neck tenderness.
I removed the internal jugular vein from the mastoid process
to the clavicle.   It was so utterly collapsed, owing to the re-
formation of the thrombus above, that its anatomic identity
was extremely difficult to establish.   It was not much larger
than the pneumogastric nerve.   After its removal I again re-
moved the sigmoid thrombus and established hemorrhage from
the torcular direction.   The wound was then closed and drain-
age maintained.   The next day the temperature was 104°
and the pulse 150, but from then on they steadily decreased,
and July 6th she left the hospital entirely well, with a hearing
of seven inches by Dr. McBride's watch.

I report this case, as I believe that all similar cases should
be reported, as an encouragement to decisive action in bad
cases.

SOCIETY PROCEEDINGS.

NEW YORK ACADEMY OF MEDICINE.

SECTION ON OTOLOGY.

Meeting of October 12, 1912.

Paper: Case of Congenital Bilateral Microtia With Total Osseous Atresia of Both External Auditory Canals.*

By JOHN RANDOLPH PAGE, M. D.,

NEW YORK.

DISCUSSION.

DR. KERRISON said that he had had an opportunity of seeing this patient before the operation and also of observing the operation. He had also seen the result and discussed with the mother the effect upon the hearing. There seems no doubt that this little patient now had a useful degree of hearing which was surely the result of the operation.

DR. BLODGETT said that inasmuch as the mastoid cells were found to be full of mucopurulent secretion at the time of the operation, he would now ask (as it was proposed to operate on the other ear) whether there were any signs of this condition in the ear first operated, for if so, it might be a guide to the condition of the mastoid cells in the other ear.

DR. BRYANT said that Dr. Page was to be congratulated upon the result of his operation. The result was as good as he had ever seen in a case of this kind. The results obtained in Dr. Page's operation are so eminently satisfactory that they furnish a strong incentive to operate on children afflicted with this deformity. Heretofore children with this type of deformity have not received proper attention, and have been allowed to grow up with defective hearing.

*See page 1079.

DR. JOHNSON inquired whether there was any evidence of any inflammatory process about the mastoid region before the operation.

DR. PAGE said that the boy had showed no evidence of having mucopurulent material in the mastoid and middle ear. Perhaps the letting out of this thick material had aided his hearing. The only thing he could discern in the X-ray picture was a very dark shadow over both mastoids; what Dr. Cole took to be a tympanum in the picture was very indefinite to his untutored eye. Both mastoids looked very dark. This was probably due to the pus. The child's mastoid region looked perfectly normal, and there was no redness or appearance of inflammation. If there had been any, it would certainly have suggested the pus.

Interesting Features of Chronic Middle Ear Suppuration.

DR. W. SOHIER BRYANT presented a case of chronic middle ear suppuration. The case showed in a very striking way the results of former methods, and, by contrast, showed what results our present technical knowledge can produce.

The results of former treatment were considerable deafness, loss of tympanic structures, chronic suppuration, and necessity for radical mastoid operations, sometimes unfortunately resulting in persisting chronic suppuration.

The history of the patient.—The patient twenty-one years ago had an abscess in the left ear. This ear has discharged varying amounts ever since. Very fetid discharge. Two years ago had very severe attacks of dizziness.

Inspection.—Left ear half full of greenish very fetid fluid. General destruction of tympanic contents and membrana tympani. Granulations hanging from the tegmen tympani and antri. Watch heard one and one-half inches in left ear.

Left ear cleaned out with alcohol. Patient instructed to use 1/2,000 bichlorid of mercury syringing.

On second visit, fifth day, left ear sweet, no fetor. Granulations shrunken.

On third visit, ninth day, fundus of left ear damp and a little purulent; granulations, a trace.

On fourth visit, twenty-second day, ear entirely dry and clean.

The patient illustrated another important point, namely, the

possibility of healing old chronic middle ear suppuration by a little antiseptic care.

Even the presence of fetor, granulations, and carious bone, is no contraindication for a good prognosis.

In cases of acute purulent inflammation, as well as in cases of chronic purulent inflammation of the middle ear, operative interference is indicated in many more cases than is generally recognized. These indications should be carefully studied, and if they are followed the operation would be performed in time to prevent the destruction of the tympanic structures. The result would be the maintenance or restoration of good hearing, according to the case. In other words, early operating should preclude and should prevent the loss of any tympanic structure which would lead to a loss of hearing.

Many of the papers read at the Ninth International Otological Congress, held in Boston last August, dealt with this important subject. Reports given at that meeting showed that cases of purulent inflammation of the middle ear offer a very good prognosis after early operative interference. It was shown, further, that in cases not likely to come to a favorable convalescence by disinfecting methods, chronic middle ear suppuration could be immediately checked by early operation on the antrum.

What is to govern our determination in the selection of operative treatment? The chief determining points lie in the osseous structure of the mastoid antral region. Suppuration is usually persistent in cases where the region about the antrum is not cellular and pneumatic; whereas in cases where the antral bone is cellular, the suppuration runs a short course.

DISCUSSION.

Dr. Johnson asked that Dr. Bryant give a little fuller description of his method of treating these cases.

Dr. Herzog said that during the past five years it had been his custom to pursue the following treatment in chronic purulent otitis media when there was a fair sized opening in the membrana tympana and no caries: First, he would thoroughly cleanse the ear of secretion by mopping with dry absorbent cotton; then have patient lie down and put ten drops of ninety-five per cent alcohol into the external auditory meatus, leaving it in for five minutes; then, after thoroughly drying the ear,

he applies five per cent solution of potassium permanganate.
This treatment pursued every three to four days results in a
permanent cure in a reasonable time.

Dr. Voislowsky inquired as to Dr. Bryant's percentage of
cure in cases treated by this method.

Dr. Scruton said that a few years ago he had seen a paper
by Dr. Bryant reporting a large number of cases of chronic ear
suppuration cured by boric acid powder. He would like to
know if the doctor still continues this treatment, or whether it
had been discarded, or whether it was applied to a different
sort of case.

Dr. Bryant said that in treating cases of chronic middle ear
suppuration he wipes out the middle ear as clean as possible at
the first visit of the patient, and orders alcohol instillations to
be used at home by the patient. In extremely foul cases he or-
ders the patient to syringe the ear at morning and night with a
solution (1/2000) of bichlorid of mercury. This treatment is
to be continued for seven to ten days. At the end of this time
the ear is generally clean, the granulations gone, and the treat-
ment is discontinued or changed to insufflations with boric acid
or xeroform powder. No operative work is needed.

Furthermore, Dr. Bryant said that he wished to emphasize
the point that cases of chronic middle ear suppuration should
be treated before the destruction of the tympanic structures.
In cases of acute mastoiditis, or in cases where, in middle ear
suppuration, there is a sclerosed mastoid, the operation on the
antrum should be performed, for in these cases a chronic sup-
puration is likely to develop. By draining the antrum through
the mastoid process the suppuration will cease.

Dr. Bryant said that he was sure that we are warranted in
operating on a purulent ear earlier than has been customary in
the past. We should operate early, not only in cases where
there are signs of acute mastoiditis, but also in cases where
there is purulent infection of the middle ear and where the
skigram shows an absence of air cells in the neighborhood of
the mastoid antrum.

Dr. Kerrison: Just a simple mastoidectomy?

Dr. Bryant said that he did not know what percentage of
his cases of chronic suppuration were cured by antiseptic treat-
ment. He thought it was above 90 per cent in all cases. Cases
of long standing suppuration are especially amenable to anti-

septic treatment, since the drainage in these cases is usually better than in more recent cases. If, in these cases, good drainage is established and local resistance improved by cleansing the walls of the bony cavity, the cases usually heal permanently.

As to the use of boric acid powder, Dr. Bryant said that he usually used it, but that the case presented was so fetid and foul that the powder would have been of no service, and he used the bichlorid of mercury on account of its greater bactericidal power.

Paper: Sinus Thrombosis—Grunert's Operation—Recovery.*

By FRANK TUCKER HOPKINS, M. D.,

NEW YORK.

DISCUSSION.

DR. EAGLETON asked if he was correct in understanding that the jugular vein was tied at the first operation.

DR. HOPKINS replied that it was a simple mastoid operation. Later there was the operation on the sinus and jugular, and then the Grunert operation.

DR. EAGLETON said he thought that every one was now using the Grunert operation in these cases. Grunert's monograph on the exposing of the sinus and jugular vein, to his mind, put an entirely different aspect on cases of sinus thrombosis, and when one has once applied the technic of going into the jugular by following the groove, there are few difficulties encountered. He had thought that all the men did this as a routine practice in cases where there was not free bleeding from below, as well as tying the jugular. The most satisfactory cases he has had of sinus thrombosis have been those in which there was no clot high up, but with profound sepsis. Where there was a clot down in the jugular, it could only be reached by going into the bulb itself. Dr. Hopkins had spoken of the danger of hemorrhage from the inferior petrosal. He had seen a case in which there was free bleeding from below the inferior petrosal, yet a continuation of the sepsis, showing that the infection was not removed. In this instance an opening was made into the jugular bulb, and the patient got well immediately.

*See page 1082.

Dr. Hopkins had also spoken of curetting. He himself thought that curettement in sinus thrombosis was a thing of the past. It had been his practice to find the clot and not try to establish bleeding below, but to keep exposing from above until there was hemorrhage from the removal of the clot, and that was only possible by making a modified Grunert.

He had never had a facial paralysis result from the Grunert operation, though he had performed it a number of times. While in Vienna, some years ago, he saw many cases of facial paralysis caused by going through the inferior wall of the meatus; this will almost invariably be followed by facial paralysis. In Vienna, in those days, practically every case of sinus thrombosis had facial paralysis. He had never seen a case of facial paralysis where Grunert's original technic was followed.

DR. BRYANT maintained that in cases requiring ligation of the jugular vein, in order to remove the infected clot from the jugular bulb, the wisest procedure is to expose the sinus as freely as possible and follow it down to the jugular bulb, keeping a sharp watch, in the meantime, to avoid the facial nerve in front. The clot is then easily removed and the passage from the upper to the lower wounds is carefully cleansed.

DR. KERRISON said he hoped the discussion would bring out the views of the members as to the indications for the Grunert operation. It was very rarely performed at the Manhattan Eye and Ear Hospital. Technically it would seem to be comparatively easy in the case of a child, but exceedingly difficult in the case of an adult patient. As bearing upon the frequency with which it may be indicated, the large series of cases operated upon in the Boston Eye and Ear Infirmary and reported by Dr. Crockett had recurred to his memory. In this series of sixty cases no mention of a Grunert operation occurred, and the average of recoveries had been as good, as, if not better than, any series reported in New York.

Dr. Kerrison said that he himself had operated on ten cases, eight of which were of children suffering from ear lesions secondary to infectious diseases. Several of these were cases of jugular bulb infection, which, however, had yielded very satisfactorily to simple jugular resection. This, as far as he had been able to see, usually controlled the infection.

DR. HOPKINS said that he knew of two cases in which the Grunert operation was performed at the Manhattan. As he

had said in his paper, in these cases the operation was done rather late. No operation should be performed unless clearly indicated. Indications are easily obtained if the septic condition of the patient continues after the operation. Especially if by obtaining the discharge from the bulb and lower part of the sinus, infection were found still present, that would seem to him to be the indication for the operation. It would hardly be an operation which one would do on a case where the sinus was thrombosed and the jugular resected. Facial nerve injury is very rare. Speaking of his first cases, Grunert reported having treated eleven in this way, and in only one was there a temporary disturbance. He himself had known of no case where there had been facial nerve injury, but this possibility should be borne in mind and the work done with care in order to avoid cutting the nerve in case it should be a little out of its natural course.

Dr. Eagleton remarked that it seemed to him that in every case in which after opening the sinus and finding a clot and the bleeding not established from below—not by curetting, but simply because the bottom of the clot was not reached—one should go down until there was bleeding. He ties the majority of these cases, but goes down into the jugular bulb when he does not get bleeding by opening the sinus in the exposed part. That was a point that Grunert made.

Paper: Facial Paralysis in Mastoid Surgery.*

By Charles W. Perkins, M. D.,

New York.

DISCUSSION.

Dr. Bryant said that he was confident that if the directions given by Dr. Perkins were followed, there would be no facial paralysis resulting from surgical trauma. The surest safeguard against paralysis is good illumination of the operative field. Good illumination prevents the operator from losing his way, and if the facial nerve is accidentally exposed, it will be recognized at once and further damage prevented. The twitching of the face is a very unreliable symptom of threatened damage to the facial nerve.

Dr. Scruton mentioned a case of double simple mastoidecto-
my resulting in complete double facial paralysis. The patient,
a syphilitic subject, showed a partial involvement of the facial
nerve on the right before operation. Paralysis was not com-
pletely bilateral until about ten days had passed after opera-
tion.

Another reason for injury to the facial nerve is that most
assistants use sharp pointed forceps to handle the gauze
sponges. The sharp points frequently work through the mesh
of the gauze and are then in a position to damage delicate
structures like the facial nerve. Recommend the use of a dull
mastoid curette of suitable size for pressing gauze sponges into
the mastoid cavity or middle ear during operation.

Dr. Berens has a specimen showing the facial nerve imme-
diately under the cortex in the posterior bony canal wall, in-
stead of being in the usual position at the level of the external
semicircular canal. Injury to the nerve would be almost a cer-
tainty if such abnormal condition existed at an operation.

Dr. Voislowsky said that he never read a paper on surgery
of the facial canal or heard it spoken of without being remind-
ed of a case he saw in 1899 or '90, and this may be the case to
which Dr. Scruton had referred. This was a skull which Dr.
Berens had borrowed from Dr. Thompson, with the intention
of demonstrating it to some students, and during the demon-
stration he came upon this anomalous facial nerve. As Dr.
Scruton had described it, it came down posteriorly. The speci-
men was now in the possession of Dr. Berens. If that ear
had been operated upon in the living subject, it would hardly
seem possible, with the best light and most careful sponging,
for the man operating to have avoided having a facial
paralysis.

Dr. Robinson said that there was very little to add to what
Dr. Perkins' had said. He would like, however, to call atten-
tion to the aid that the anesthetist could give by constantly
watching the patient's face during the operation to notice the
faintest twitch.

Dr. Hays told of a man who some years ago had remarked
that no one who understood anatomy well would ever have a
case of facial paralysis, and the very next day operated, with
a resulting very serious facial paralysis. He himself had had
a very serious case of facial paralysis in the infirmary. The

patient was a woman who had suffered from mastoiditis for six weeks, and had complained of some slight twitching on one side. At the time of the operation no exposure of the nerve was made, and there was no twitching. The second day after, however, she developed a total facial paralysis which is very distressing and is growing worse all the time. He had been considering the advisability of suturing the hypoglossal nerve to the facial, and would like to know if any one had had any experience of this kind.

DR. BLODGET said that Dr. Hartley claimed that he would be very glad to operate any case of facial paralysis, with a reasonable hope of curing it, if it was not of too long standing.

DR. HOPKINS said that he had reported a case of superficial nerve paralysis which occurred at the first blow of the chisel on the cortex. By very good fortune, the first blow uncovered the nerve but did not injure it. The nerve was disturbed for a short time, but there was no permanent damage.

DR. PAGE, referring to the twitching of the face from irritation of the nerve with an instrument, told of a case upon which he had operated some time ago. The patient was a young man, a diabetic, and had a very extensive mastoid involvement. He operated as rapidly as possible because of his condition and was looking for the aditus. There was a very large cell, which he took to be the antrum, but which was not. The dura was very much higher than usual, and this misled him, so in going forward to find the aditus he ran into the facial nerve, just below the horizontal canal, and injured it. He had told the anesthetist to watch the patient's face from the beginning, and no evidence of twitching was noted. As soon as he discovered that he had injured the nerve, the patient was allowed to come from under the anesthetic to make sure that the nerve had been injured. In this case he had made a great mistake in not at once covering the nerve with cargile membrane to protect it, and to prevent, if possible, the ingrowth of connective tissue at its injured portion.

DR. EAGLETON said that he was glad to hear that Dr. Hartley was so optimistic in regard to curing facial paralysis. He had seen a very eminent neurologic surgeon search for three hours trying to find the end of the facial nerve. He has now under observation a case which was operated upon by a neurologic surgeon, and while the patient claims to be very much better,

there is still what he would regard as a very bad facial paralysis. He had seen two cases operated upon by Dr. Krause of Berlin, and while he claimed that in a few months they would be very much better, at the time he exhibited them they were not very greatly improved. In all the cases of anastomosis that have come under his observation, the results have not been such as to lead him to think it was of great advantage In Dr. Perkins' very fine paper, no mention was made of one point which had been of great value to him—that is, the use of Dr. Richards' cutting curette. He has never been satisfied with his radical operation, but he never performs one without realizing the value of this curette. Although he has exposed the nerve a number of times, he has never injured it. The curette is very much safer than the chisel. If the nerve is exposed in this way, it is easily recognizable, and with ordinary care no harm is done. A year ago, while dissecting some bones while studying the labyrinthine operation, he met with a condition in the temporal bone which would explain why we have facial paralysis in acute mastoid, sometimes before the operation. While the sinus tympanicus has been very well described, we do not always remember that sometimes, perhaps in a majority of cases, there are several small cells, which may extend under the facial and become continuous with the mastoid cells. In this case, he was amazed to find that he could pass a probe from the mastoid into the tympanic cavity under the facial nerve. If that case had had an acute mastoid it would have had paralysis the second or third day, for the whole of the bridge was undermined. He had never heard this spoken of as being the cause of the facial paralysis which develops in acute mastoid without operation, but this specimen made it very plain that it could be the cause.

Dr. KERRISON said that Dr. Perkins had outlined an excellent set of rules for minimizing the danger of postoperative facial paralysis. No set of rules, however, could absolutely ensure against this accident—particularly the paralysis which comes on gradually, i. e., a few days after the operation. He recalled a trying experience of his own in operating upon a baby eighteen months old. This patient recovered from the anesthesia with complete facial paralysis. These symptoms began to subside within a week and inside of a month recovery was complete. In spite of every care there is likely to be a cer-

tain number of cases of temporary facial paralysis. Their
number, however, should be small, and with care permanent
paralysis should not occur.

DR. PERKINS said that he was referring to injuries of the
nerve which resulted in permanent facial paralysis, and not of
cases of neuritis, frequently from causes which cannot be avoid-
ed. These, as Dr. Kerrison had said, frequently clear up and
are not serious. He had mentioned Dr. Richards' curettes
and had found them very valuable instruments.

ABSTRACTS FROM CURRENT LITERATURE.

Congenital Syphilitic Deafness Treated by Salvarsan.

GEORGE NIXON BEGGS (*Brit. Med. Journal*, August. 1912, p. 348) finds salvarsan only helpful in cases of syphilitic deafness when combined with inunctions of mercury and large doses of potassium iodid given internally. He reports one case illustrating this. *Richards.*

Otalgia Angiosclerotica.

CONRAD STEIN (*Wiener klin. Woch.*, June, 1912) thinks many socalled neurotic neuralgias of the ear (all other causes for the pain being excluded) are due to an arteriosclerosis of the blood vessels supplying the ear. He suggests the name angiosclerotic otalgia for the condition. He has found the use of diuretin to give good results; 0.5 grms. ($7\frac{1}{2}$ grs.) is given three to four times daily, and for several weeks. *Richards.*

Indications and Contraindications for the Use of Salvarsan in Aural Syphilis.

OSCAR BECK (*Muenchener medizinische Wochenschrift*. August, 1912). If the inner ear is in normal condition there is no contraindication to the use of salvarsan. In old syphilis of the ear its use should be guarded, especially if there be degeneration of the cochlea or vestibular nerve. In congenital deafness it may be of value if injected while the child is still young, provided the hearing for ordinary speech is at least four feet, and the vestibular apparatus functionates normally. If syphilis of the ear is suspected, the advice of an otologist should be had before the injection of salvarsan. *Richards.*

Meningitis as a Cause of Deafness.

J. KERR LOVE (*British Medical Journal*, August 24, 1912) says there are now more than 5,000 children in the special schools for the deaf. Of these, one-fifth were due to meningitis. He urges the study of all cases of acquired deafness, in order to find out how much true hereditary deafness there is.

The hereditary deaf ought to be taught in regard to the danger of marriage and intermarriage. He urges the appointment of ear surgeons to all fever hospitals, since acute exanthemata are prolific causes of acquired deafness and frequently lead to fatal complications.   *Richards.*

Qtosclerosis and Allied Conditions.

ALBERT A. GRAY (*Laryngoscope,* January, 1912, p. 1) considers as clinical symptoms in otosclerosis deafness of insidious onset, relatively prolonged bone conduction, a loss of hearing for the lower notes, and a normal position of the tympanic membrane. These symptoms are often accompanied by the complaint of paracusis and tinnitus, and sometimes there is a rosy tint in the region of the promontory. The author presents five slides which illustrate the changes due to these symptoms in the bony capsule of the labyrinth, which has its seat usually in the region in front of and above the oval window, and leads to the bony fixation of the stapes.

Otosclerosis in a large number of cases is hereditary. Koerner makes the suggestion that otosclerotics should not marry, since children of otosclerotic children are more liable to this disease than those born of normal hearing parents. Local and constitutional conditions often determine the onset and course of otosclerosis in individuals who inherit the tendency to this disease.   *Richards.*

The Consideration of the Mechanism of Pressure in the Production of Vertigo.

CLARENCE JOHN BLAKE (*Boston Med. and Surg. Journal,* September, 1911, p. 469). In a series of cases of recurrent vertiginous attack where there was insufficient intralabyrinthine pressure, or in which the extrinsic pressure factor was at a minimum or absent, lumbar puncture has given temporary relief from this condition, and in the majority of cases, entire relief from the vertiginous attacks during a period of from a few months to three years. In no instance was there a more severe consequence than severe headache of two or three days' duration. In every case 10 to 15 ccm. of fluid was withdrawn and the patient closely observed for a few days. Oftentimes the lumbar puncture is done in preference to immediate incudectomy, stapedectomy, or an operation upon the vestibule.
                          Richards.

Vertigo of Vestibular Nystagmus.

PHILLIP D. KERRISON (*Laryngoscope,* October, 1911, p. 978) defines two distinct types of labyrinthine vertigo and associated ataxia—one the familiar spontaneous type, due to vestibular irritation and occurring only during the acute stage of suppurative labyrinthitis; and the other not spontaneous, not constant, not necessarily accompanied by nystagmus, characteristic only of the latent stage of the disease, and induced solely by sudden and unexpected calls upon the lost or defective orientation sense, in the maintenance of which the intact vestibular organs are so important a factor. He infers from his experiments that in cases of impaired orientation, especially where the sight is lost or where one vestibular apparatus has been destroyed, that the muscular, arthroidal and tactile impressions of space and position become more sensitive and accurate, and until these senses are so developed as to make up for the lost factor or orientation, the patient suffers from attacks of vertigo or vestibular paralysis.'   *Richards.*

Vaccines in Suppurative Otitis Media.

ARTHUR C. CHRISTIE (*Medical Record,* September, 1912), after three years of experience with vaccines, finds them especially useful in those cases which are resistant to local treatment. When it becomes evident, after ten days to three weeks of local treatment, that the case will be one of long duration, the use of vaccines is begun, the local treatment being continued, since they are not used to the exclusion of other methods of treatment. Out of twenty-five cases treated with vaccines, in twenty the vaccines were entirely successful. These twenty-five cases were all of severe type, and most of them had persisted after long continued local treatment.

All cases of acute otitis media which do not end fatally are curable theoretically at least, but the large number of patients with chronic otitis media prove that many are not cured, but become chronic, and those cases which are not curable by local measures or the vaccines should have the simple mastoid operation performed.

In subacute cases vaccines are also of value, but in chronic cases their field of usefulness is much more restricted.

Richards.

A Case of Otosclerosis With Pathology.

ALFRED M. AMADON (*Laryngoscope*, July, 1912, p. 927), from his study of otosclerosis, finds a little more than 20 per cent are hereditary, while Denker finds 40.5 per cent, Lucae 37 per cent, and Siebenmann 35 per cent. Heiman has found only 9 per cent to show clear heredity. It has been noticed that there is tendency to drop the inheritance after the second or third generation, but never does it skip a generation from its appearance until its disappearance. It is very often associated with anemia, arteriosclerosis, or some of the arthritides. The otosclerotic processes seat themselves in the bone of the labyrinthine capsule, on account of its peculiar anatomic and histologic characteristics. It is the author's opinion that the capsule of the labyrinth takes its place along with the other parts of the bony skeleton that are, for anatomic and physiologic reasons, peculiarly liable to pathologic changes as the result of some more or less general condition, like the disturbance of nutrition. That the resulting metaplasia presents unique features is not due to any specific and local existing cause, but to a unique normal histology. At present, prognosis as far as treatment is concerned is hopeless. If the process can be checked before there is any anchylosis or break in the continuity of the annular ligament, or if the process can be made to stop short of the affection of the cochlear structures, as much will have been done as is physically possible.   *Richards.*

Epithelial Grafting as a Means of Effecting a Sure and Rapid Healing of the Cavity After a Complete Mastoid.

CHARLES A. BALLANCE (*Lancet*, August, 1912, p. 428) employs epithelium grafting in all his cases of mastoid operation whether they are of a suppurative or nonsuppurative condition. The graft may be applied at the close of the operation, but ordinarily is done seven or eight days after the mastoid operation. The operated cavity is thoroughly cleaned with hydrogen peroxid and warm sterile saline solution. The graft, very thin and preferably from the thigh, is placed in such a manner as to cover the tegmen, the inner wall of the attic, tympanum and antrum with epithelium. Any air or blood separating the graft from the inner wall of the tympanoantral cavity is sucked out beneath the edges of the graft by a pipette. The graft is held in place against the bone by a small sterile strip of gauze dusted

with aristol. Then two grafts are so placed as to cover the raw edge of the posterior margin of the meatus, and the mastoid flap is replaced and sutured with fine silkworm gut, and a dry sterile dressing applied. The external dressing is changed daily, and the gauze tampon from the third to sixth day. Subsequent treatment consists of irrigating twice daily with hydrogen peroxid and rectified spirits, or dry tamponing. He reports that 75 per cent of all his cases completed by the graft method have healed within two or three weeks and have improved in hearing.   *Richards.*

The Value of Lumbar Puncture in the Treatment of Aural Vertigo.

JAMES J. PUTNAM (*Boston Med. and Surg. Journal,* September, 1911, p. 472). The best cases responding to treatment by lumbar puncture are those where the various tests show the labyrinthine apparatus in a fairly normal condition. Where the neural degeneration is far advanced, the effects of withdrawal of cerebrospinal fluid are less marked at the outset, and produce less and less results, and are finally of no benefit.

The most favorable cases are those of pure labyrinthine origin and of relatively short duration. In such cases no great disturbance of hearing is present, and especially no middle ear disease. In these cases the sensitiveness of the labyrinthine ap-repetitions of the operation, which in the better cases work well, paratus to the galvanic current, which before the treatment by lumbar puncture may have been poor, as shown by the fact that it required a strong current (even 20 ma. or more) to cause the vertigo, nystagmus and inclination, even if it could be induced at all, may become again normal after the puncture.

About 15 to 20 ccm. of fluid is withdrawn, and the relief from vertigo sometimes occurs at once, and sometimes after several days. In a fair proportion of cases, any tinnitus which accompanies the vertigo, and oftentimes the deafness, is relieved. Severe headache of one or two days' duration is the only bad result from the lumbar puncture.   *Richards.*

The Technic of Auditory Examination in Infancy.

P. MAURICE CONSTANTIN (*Laryngology,* July, 1912, p. 353) regards the examination of children for deafmutism difficult. The Escat tests give the most satisfaction in testing children

of two years and under, with a moderate amount of intelligence. An aerial test is made by calling in a loud voice, and from behind, the name of the child, followed by tones such as produced by the pronunciation of "bonbon" and "zizi." If the child in any way responds, the test is made in a whisper For instrumental examination a deep ut^2 fork is used, with Gradenigo's index. The fork is placed toward the ear opposite the observer, and then at the other ear, and each tine made to vibrate up to its maximum index. The child inclines its head in whichever direction he first hears the fork. The ut^6 fork of Lucae, or the sol^6 (G^4) of Quix, provided with a percussive hammer, or the Galton-Edelmann whistle, can be used. The child usually sits on the lap of the assistant, and the testing instrument is held behind the child, thus avoiding drawing his attention to it. During the examination the rotation of the head or the deviation of the eyes to the more stimulated side, and the facial expressions are carefully noticed by the observer, and indicate the child's hearing. A watch that can be stopped and started at the observer's inclination gives perhaps the best results for bone conduction, the eyes deviating toward the side of the ticking watch. Two tuning forks, one vibratory, one not, can be used. The child will incline head toward the vibrating one. *Richards.*

Causes Leading to Educational Deafness in Children.

MACLEOD YEARSLEY (*Lancet,* July, 1912, p. 138), from a study of 1076 children born with congenital deafness, reports heredity and consanguinity to be the most prominent factors, with a possibility that alcohol, insanity, and perhaps syphilis are the most important of minor cases. Out of 123 families, deaf birth occurred in the direct line or in collateral branches in 56, or 45.5 per cent; and out of 309 families, consanguinity of parents was present in 22, or 7.08 per cent; whilst the percentage of parental relation in 592 acquired cases was only 0.32. The prohibition of marriages of deaf-born and of blood relations, and the union of alcoholics, syphilitics, and those having a family taint of insanity, epilepsy, or other neuroses, is strongly urged to prevent this deafmute condition.

Of 592 cases of acquired deafmutism, 74.2 per cent were due to suppurative or catarrhal middle ear disease, and of this number 21.9 per cent could have been avoided if treated in

time. Infectious fever was a causative factor in 26.3 per cent, and diseases of the nervous system, such as meningitis, is responsible for 10.9. Adenoids are distinctly a cause of deafness, and the parent should be made to understand their danger to the child's hearing and respiratory tracts. Parents and general practitioners are urged to care and look out for the ears, especially discharging ears, in very young children, and to regard the ear as an important organ of the body, needing prompt and energetic attention to prevent disease and resultant deafness.   *Richards.*

Prominent Ears.

ROBERT T. MORRIS (*Med. Record,* March, 1912, p. 561). This operation is only advised when the ears are so prominent that they cause deflected currents of cold air to irritate the drum membrane, or when they cause the owner mental agitation.

The author describes his technic thus: Take an ellipse of skin, one-half from the mastoid region of the scalp and one-half from the posterior part of the ear, first determined by pressing the ear flat against the mastoid. Grasp the ear by the helix, drawing it well forward, and snip away the ellipse of skin with curved scissors, leaving the subcutaneous tissue as intact as possible. Tie the branches of the posterior auricular and superficial temporal arteries with catgut threaded in a needle if necessary. Outline with small curved scissors the cartilage to be removed, and then gradually cut away the cartilage from the skin of the anterior aspect of the ear by alternately pressing and snipping. Make one or more openings in the concha and antihelix for proper drainage, if this has not already been done, by buttonholing in the removal of the cartilage. To prevent blood clotting upon the drum membrane, pack the external auditory meatus with absorbent cotton previous to operating. Careful dressing is important. Fit two thicknesses of iodoform gauze, cut to the shape of the wound, between the skin of the helix and the skin of the scalp. Pack iodoform gauze upon the skin of the anterior part of the ear and to the fossa of the helix, compressing the skin nicely and evenly, thus preventing blood from collecting beneath it. Leave the meatus open or loosely cover with gauze.   *Richards.*

Dangers Incurred as a Result of the Nonrecognition of Acute Aural Infections, With Statistical Data.

F. E. KITTREDGE (*Boston Med. and Surg. Jour.*, September, 1912, p. 358).   From a study of the vital statistics of New Hampshire and the relation between the number of cases of mastoid operation performed and the number of cases of death which was reported from meningitis, Kittredge comes to the conclusion that the more frequent recognition of suspected middle ear trouble with mastoid involvement, with operative relief for the same, would lessen the number of deaths reported as from simple meningitis.   He is of the opinion that if the recognition of suspected conditions in the middle ear was more frequent, and as prompt response made to the imperative demand for surgical intervention as is given in acute appendix cases, there would be a much smaller number of deaths reported from simple meningitis.   It should be impressed upon the profession that dangerously infected mastoids may be present without presenting the usual symptoms as formerly taught—that swelling posterior to the ear is only present late in the course of the disease, that marked tenderness usually subsides with acute inflammatory symptoms, that the temperature chart is not always a safe guide, a temperature nearly normal not necessarily meaning absence of danger, while a high temperature should cause one to suspect some serious complication.   Even the absence of free pus in the mastoid cells does not necessarily mean that the case has been operated upon unwisely, since there may be present in the mastoid antrum, scattered throughout the cellular structure, highly infected granulations.   *Richards.*

Bacteremia and Purulent Ear Discharge.

J. H. GUENTZER (*Laryngoscope*, July, 1912, p. 953) reports from a series of blood cultures made in forty-nine cases of acute and chronic middle ear suppuration.   Five showed bacteremia; four had streptococci, and one had staphylococci in the blood.   Eight of the remaining cultures were contaminated, and thirty-five sterile.   Out of these five cases, two had no apparent intracranial involvement, yet showed a streptococcemia on blood culture.   Duel and Wright reported a series of fifty-seven cases of blood culture, two of which were in frontal sinus cases and fifty-five in middle ear cases, and of

this group fourteen showed a positive bacteremia, and of this number, nine cases were of mastoiditis complications, all of the patients recovering after operation without any intracranial sequelæ.   It has been shown that bacteremia occurring during a purulent middle ear disease, of and by itself, without taking into consideration the clinical symptoms and objective manifestations that are observed in a sigmoid or lateral sinus thrombosis, cannot be taken ipso facto as indicating this condition of thrombosis, nor can it alone be taken for a pathognomonic sign, and therefore when found be indicative for operative interference within the sinus precincts. Neither does a bacteremia in purulent middle ear diseases indicate the presence of an infected thrombus in the lateral or sigmoid sinus, the thrombus, if present, most likely remaining sterile till a late stage.   During this late stage the thrombus, by disintegration, may cause distal metastasis; but before this late stage the microörganisms have already reached the circulation by a different way.   .   *Richards.*

The Influence of Diseases of the Ears on the Point of Coagulation of the Blood.

ERNST URBANTSCHITSCH, Vienna (*Monat. für Ohrenheilkunde,* September, 1912).   As a result of a very careful investigation of the coagulability of the blood in fifty-five cases with intracranial complications and many normal patients, the author has been able to draw some very important conclusions which will be a great addition to our very meager knowledge of the differential diagnosis between sinus thrombosis and pyemia.

Wright's method of determining the coagulability of the blood was used, and seems to be the one on which the greatest reliance can be placed.   The principal objection is the cost, as the pipettes, which can only be used once, cost in Germany about ten cents.   A description of the technic can be obtained in many English textbooks, but if one intends to follow out this line of research, the original work of Urbantschitsch should be consulted.

His conclusions are as follows:

1. We possess in Wright's procedure a method of great exactness, which can be estimated in seconds, and which is

the only known method by which these small differences can be estimated.

2. All cases of pyemia show marked slowing of the coagulation time.

3. No differential diagnosis between complete or wall thromboses is possible by this method. It is impossible to differentiate whether the thrombus is infected or not.

4. Shortened coagulation time is not pathognomonic for thrombus. Other intracranial complications, such as brain abscess, also show shortened coagulation period.

5. All cases of sepsis show normal or somewhat lengthened blood coagulation period.

6. The value of the determination of the coagulation point of the blood is of value in otology: (a) For the differential diagnosis between pyemia and sepsis, and (b) on account of the possibility in uncertain cases to shut out the possibility of a thrombotic process.

7. Medication which, will influence the blood coagulation point will not influence the pyemic process.   *Horn.*

Vaccines in Aural Practice.

C. ERNEST WEST (*Clin. Journ.*, October, 1911, p. 44). Cases of furunculosis of the meatus generally clear up after an injection of forty million killed staphylococcus aureus, followed in from three days to a week by a similar dose, and at the end of the third week by one hundred million. For recurrent furuncles, an isolated injection of forty million is required once in six months.

In treating cases of lateral sinus thrombosis, the patient, before leaving the operating table, receives a serum of 40 cc. of pyogenes, followed by a similar dose twelve hours later. In addition, a vaccine from a stock streptococcus pyogenes, in doses of five million, is given. This stock vaccine is later substituted by an autogenous vaccine, the dose increasing to 100 million, if necessary. Seventy-five per cent of these cases recover.

Meninges and brain infections are helped by the sera and vaccine only when the case is diagnosed early enough and the primary infections removed.

In chronic suppurative otitis media the most marked results have been obtained from vaccine in pyocyaneus infections.

In the treatment of nonsuppurative conditions, originating
in the nasopharynx, vaccine has greatly lessened postnasal
catarrh, but deafness remains the same. Cultures taken from
the nasopharynx show in a series of fifty consecutive cases
70 per cent pneumococcal. In a second group of cases
staphylococcus aureus were dominant in 26 per cent; staphylo-
cocci were present in over 40 per cent of the cases. The
streptococci bulk less largely, and as the series included only
strictly chronic cases, catarrhalis was rarely present (8 per
cent). Other Gram-negative cocci were present in large
number in 14 per cent of cases. The only other group of
importance is furnished by infections by Friedländer's bacillus.
In pneumococcal cases, autogenous and stock pneumococcal
vaccines carried up to large doses have given no results what-
ever. In cases of mixed infection the pneumococcal element
survive, but the Friedländer organism disappears. The
staphylococcal cases show improvement in catarrh, and the
catarrhalis and Friedländer cases do well. In streptoccocal
cases the initial dose of pyogenes is about five million. The
repetition and increase of the dose depends upon the individ-
ual and the results obtained.

In these cases no attempt was made at control by the opsonic
index, nor are more elaborate methods considered to be neces-
sary.   *Richards.*

Acute Suppurative Otitis Media.

ROBERT H. WOODS, Dublin (*British Med. Journal,* August
24, 1912), considers the process in acute otitis media to be
initially a monoseptic one, and the one thing necessary to
bring about a cure is to prevent polysepsis. His technic is to
remove the discharge from the meatus by efficient syring-
ing with slightly alkaline sterile water, repeated as often as
discharge appears at the external meatus. After the syring-
ing, the ear should be dried. The meatus is then filled with
some antiseptic, either in the form of a lotion, such as boro-
alcohol, which is a saturated solution of boric acid and 40 per
cent alcohol, or with boric acid. Peroxid of hydrogen should
not be used. Sodium salicylate is the best drug to use inter-
nally.

After the membrane is bulged, a linear incision should be
made in it behind and below the handle of the malleus. Pain
subsides, and the temperature drops in a few hours, as a rule.

If this does not happen, it should not be taken for granted that the wound in the membrane is too small, but rather that the tympanic cavity is not the part of the temporal bone chiefly affected, but that the mastoid is probably involved. It should be opened and proper drainage provided.

If doubt exists as to the health of the meninges, the middle and posterior fauces should be opened, a perfectly safe procedure if no violence is done to the dura. Many children have signs of cerebral irritation which disappear when the drum membrane ruptures or is incised.

Rundle advises strongly against the removal of tonsils and adenoids during the early stages of any of the infectious diseases of childhood.

Alexander has found that in the exanthemata, urotropin is very useful in the prevention of ear complications. Large doses must be given. Children, one-half gram doses twice daily; adults, four to six times daily. Before mastoid operations urotropin may be given as a prophylaxis against postoperative meningitis.   *Richards.*

The Applied Anatomy of the Eustachian Tube.

J. A. BACKER (*Laryngoscope*, January, 1912, p. 21), in his study of the eustachian tube, finds the tube in a 27 cm. fetus short, relatively wide, without an isthmus, and as the middle ear is placed horizontally, and is only 2 or 3 mm. in height, its vertical diameter is equal to the height of the middle ear, so it drains the middle ear from the bottom, and drains it perfectly, as in animals. In the 32 cm. fetus, the tube has the same characteristic, but is longer and larger, and since the middle ear has increased in height rather more than the tube, it is not quite as perfectly a direct continuation of the whole middle ear as it is in the smaller fetus. The canal for the tensor tympani is beginning to show in this fetus. Before birth the tube leaves the middle ear as a cylindrical tube and maintains its caliber throughout the length, whereas at birth the opening of the tube comes off like a funnel, with its mouth toward the middle ear, instead of like a cylinder. The two are inseparably merged and form a cone which slopes inward more sharply at its base.

The chief characteristic of the adult tube is that it leaves the middle ear above the floor. The tube meets the anterior

wall of the middle ear at approximately a right angle, and there is a jumping off of, on the average, 7 mm. from the floor of the tube to the floor of the middle ear. The opening, while still funnel-shaped, is less so than at birth. The lumen is constricted by an isthmus. In ten specimens the maximum distance from the isthmus to the pharyngeal orifice was 37 mm., the minimum 17 mm., and the average 27.4 mm. The maximum length of ten tubes was 44 mm., the minimum 33 mm., and the average 37.8 mm The curvature of the adult eustachian tube as given by fifteen casts is that of an arc with a radius of 2 cm. The limits of variation are 2.5 and 1.6. Only two of the fifteen were curved on a radius of 2.5 cm. There is a marked difference between curettes made on this curvature and those of Yanakuer, which are curved on a radius of 2.5 cm. According to the specimens, a curette for the eustachian tube should have sharper curvature than those of Yankauer and Yearsley, in order to hug the external wall of the tube and so keep away from the thin bone of the internal wall which separates it from the carotid canal.

Richards.

A Severe Case of Vertigo.

MACLEOD YEARSLEY (*Lancet*, February, 1912, p. 428) reports an interesting case in which the labyrinth was destroyed with cure to the vertigo. A woman, thirty-one years of age, had suffered from repeated attacks of vertigo for eleven years. She was in good health except for these attacks, followed by constant vomiting and the sense of falling. In 1909, on examination, she was found to be deaf on the right side Her ear had discharged for eighteen months after an attack of diphtheria when eighteen years of age. The lower half of the inferior segment of the right tympanic membrane was thickened and opaque: the remainder was thin and flaccid, especially in the superior posterior quadrant, which appeared to be adherent to the head of the stapes. The left membrane showed inferior thickening. Nose and throat negative. Slight horizontal nystagmus on lateral deviation to either side. Romberg's test showed tendency to sway towards the right. Functional test: Weber, lateralized to the left. Acoumter: voice and whisper; Right, 15 feet, Left, nil; Rinné, Right, double negative: Left, positive: Bone conduction: Right, greater than — 20 seconds; Left, — 5 seconds. Galton-

Edelmann whistle: Right, 18,580.96 D. V.; Left, 40,000 D. V.
In the lower part of the scale she could hear no fork on the
right side until C. 256 was reached, for which she had percep-
tion only.

In March, 1910, she had had severe attacks, followed by
vomiting, weakness, and falling toward the right. Caloric
test showed marked and rather coarse nystagmus to the right.
Doses of bromid and later quinin and asafetida had no effect.
Operated upon on May 28, 1910. Right auricle turned down
by means of Lake's incision. A wide radical operation was
performed; the spine of Henle was well marked, the mastoid
being of the infantile type. The antrum was quickly reached
and found to be the size of a split pea. The bridge was re-
moved, leaving the tympanic membrane intact, which, after
the extraction of the incus through the antrum, was removed
by cutting around with a Sexton knife. The stapes, with the
tendon of the stapedius, having been taken away, the wound
was enlarged by the smoothing down of the buttress and all
overhanging projections, and a good view of the operating
field was obtained. One or two facial twitches were noted
when working near the pelvis ovalis, and the seventh nerve
was found partially exposed in that region. The ampulla of
the external semicircular canal having been opened with a fine
chisel, that passage was followed into the vestibule, which was
thoroughly curetted and swabbed out with a solution of for-
malin. The tympanic ostium of the eustachian tube was
curetted, the wound packed and the auricle readjusted. The
ear healed without complication within six weeks. During
convalescence some dizziness and nystagmus present, but these
gradually lessened and stopped. Two years later the patient
reports herself in excellent health, except for the inability to
walk straight alone after she had been walking for more than
ten minutes. She has no sense of security or locality in dark-
ness.   *Richards.*

Permeating Mastoid Meningitis.

JOHN BURGESS (*Practitioner*, December, 1911, p. 867) cites
two cases which show the uncertainty in diagnosing cases of
apparent mastoid meningitis.

Case 1.—A child of eight years and of anemic disposition
had a very severe attack of enteric fever of six weeks' dura-
tion. At the termination of fever there was loss of memory

which quickly passed off. During the fever and convalescent period her tongue was covered with several round patches of raised white fur. Five weeks later the child complained of great pain in the region of the left frontal and left ear. There were no tender spots. There was no vomiting, tache, or retraction of abdomen; temperature was normal, pulse 120 She had been eating well and was perfectly conscious, but on the previous night there had been severe pain. Phenacetin and hot stupes prescribed. Two days later, pain in left ear had been relieved by vomiting; marked paralysis of the right facial nerve; drum retracted but not injected; no tenderness over mastoid; deafness to watch and loud whispers. Next day, pain less. On the next visit temperature was 104° F. and pulse rate 128. Night reported as bad. Symptoms had every indication of otitis media or suppuration of the mastoid. Leeches were applied over the mastoid, and a fairly large dose of calomel given. For the next four days there was some improvement, the pain subsiding and the temperature returning to almost normal, although the pulse rate was still quick. On examination by an aurist the drum was found to be depressed and deafness very slight; no mastoid tenderness was present. The case was diagnosed as acute otitis media without pus formation, leaving behind it some tenderness which would quickly pass away and having no signs of meningitis. Twelve hours later general convulsions set in, followed by coma, the eyes deviating to the left. Operation was advised, but on account of no assured result, permission was refused. Death resulted.

Case 2.—A healthy girl of eight years awoke one Saturday morning with a violent headache, followed by constant vomiting. In the afternoon she was extremely restless, the headache was not so severe, but the vomiting of bile and mucus continued. She was quite sensible, temperature was 105° F. and pulse about 120. There had never been any trouble with the ears. The tongue was fairly clean. No constipation. From constant vomiting was very weak, and although not comatous, she had difficulty in fixing her attention. Monday she was very low, the hyperpyrexia continued, and she seemed to be sinking into coma. A fever specialist diagnosed the case as tuberculous meningitis with the prognosis of death within twelve hours. Previously the case had been diagnosed

as suppressed measles or scarlatina. Late Monday night the patient was very low, the pulse was uncountable and flickering, and she was barely conscious. She had every indication of meningitis, and on account of the low condition and the hopelessness of the case she was not seen again that day. On Tuesday afternoon the child was up and dressed, playing on the floor. During the night there had been a profuse discharge from the ear, after which she slept well, and on the next morning was able to be about. In this case there was no tenderness over the mastoid, nor had there been any earache or anything to point to the suppuration of the middle ear.

Richards.

Treatment of Chronic Adhesive Processes in the Middle Ear.

MACLEOD YEARSLEY AND HUGO FREY (*Journal of Laryngology*, December, 1911, p. 626). Macleod Yearsley is not very sanguine as to the results which have been and are likely to be attained in the treatment of this condition. In an accurate diagnosis from the symptoms, a physical examination and functional test is very essential, both as regards the choice of treatment and in estimating the results obtained from treatment. The relative acuity of the whispered speech and the spoken speech is a rough guide as to the prognosis, being fair when the spoken speech is better than that of the whisper, and bad when the spoken speech is worse than that of the whisper.

He thinks inflation and the use of bougies of value, and that it would be better to catheterize for a time frequently, than at longer intervals; but one must guard against overinflation and stretching of the membrane. He is opposed to autoinflation by the method of Valsalva. The conditions of the eusatchian tube and of Rosenmüller's fossa are to be studied and treated in accordance with their conditions. The correction of nasal abnormalities is to be recommended only when it is pretty evident that such correction will improve the aural condition. Otomassage is of value if used with discretion. He has not had any results with hot air or fibrolysin. The intratympanic injection of a solution of red iodid of mercury with dram vij of lanolin, made up to ounce iv with parolein, has given good results in conjunction with otomassage, even after simple inflation had failed.

Electricity has given little or no results, nor have the va-

rious operative procedures, such as mobilization of the malleus, incision of the posterior fold, tenotomy of the tensor tympani, tenotomy of the stapedius, exploratory tympanotomy, the division of adhesions, removal of portions of the membrane and removal of the major ossicles. Only in occasional cases where there are distinct and visible adhesions is ossiculectomy advised. Patients with nonoperative diseases of this kind should be taught lip reading, and the aural profession should study much more than it has done the mechanical types of aids to hearing.

Frey thinks the treatment of chronic catarrhal adhesive processes should be limited to cases in which the anatomic changes are those of hyperproduction of the connective tissue substance in the middle ear, and which clinically show a long duration and perhaps take a progressive course. They should be accompanied by visible changes in the membrane in the shape of retraction or thickening, and have the characteristics of trouble with the sound conducting apparatus, especially the lengthening of the bone conduction, when functional tests are applied. These changes often become noticeable after the process has gone on for several years, having had its first inception in acute inflammations. The pathologic condition of the septum, the middle turbinates, and accessory sinuses must be treated. There are two complaints in these cases: first, deafness; second, subjective noises. There is no specific treatment for subjective noises. What helps to influence the changes in the middle ear will help the noises, and nothing else. The deafness is due to a mechanical obstacle in the sound conducting apparatus. We therefore use all measures which tend to facilitate the mobilization of the middle ear apparatus. This treatment has to extend over weeks and months. The bougie for the dilatation of the eustachian tube often gives satisfactory results, as well as the vibratory massage of the tube with the bougie. The external application of hot air is of some value. Dintenfass introduces a small lamp directly into the meatus, and subjective noises have been reduced and in some cases have disappeared after such an application. Internally iodin is of the most value and should be continued at least six weeks in medium doses. Results are often good, and on the other hand are often failures. Pharyngoscopy by the new methods should help us somewhat by re-

moving alteration discovered by this method, heretofore undis-
covered. In reporting new methods of treatment only such
cases should be recorded which are absolutely unquestionable
as to their diagnosis, and should be recorded with their com-
plete tests before and after treatment. When this is done we
may be able to control the value of different methods on a
larger and more uniform material.   *Richards.*

Purulent Meningitis.

KOPETZKY AND HAYNES (*Laryngoscope,* June, 1912). Ko-
petzky, in a study of meningitis, comes to the conclusion that
it is a progressive disease which may terminate in a recovery
in its initial stage, or that it advances and eventually exhibits
frankly purulent characteristics which have caused the later
stage to be termed purulent meningitis. The symptoms are
dependent on increased intracranial pressure and on the
growth of bacteria and decomposition products thrown into
the circulation from the disintegration of nervous tissue. The
increased cranial pressure most often determines the outcome
in the case.

Haynes, in order to lessen the intracranial pressure, has
devised an operation for the drainage of the cisterna magna.
If the condition of meningitis is recognized sufficiently early
and relief instituted promptly, it is believed that more cases
of meningitis might be brought to recovery. The purpose
of the operation is to open the cisterna magna, without danger
of cerebellar hernia, relieve the intracranial pressure, provide
for free, but not too rapid, continuous drainage of the in-
fected cerebrospinal fluid, to avoid infection of the foramen
of Magendie, and if it be closed to reopen it, and forestall pos-
sible complications such as hydrosyphilis.

The patient is placed upon the operating table, head down,
and the anesthesia administered through nasal tubes beneath
the sterile sheet covering the patient. The incision is in the
middle line from the occipital protuberance to the spinous pro-
cess of the axis, and carried down to the occipital bone and
posterior arch of the atlas. Hemorrhage, slight and easily
arrested, is checked by mosquito clamps and the vessels ligated.

The periosteum is now stripped from the occipital bone,
taking with it the inner portions of the origin of the attached
muscles, and the occipital bone is bared for about a distance

of one and one-half to two inches vertically and an inch transversely, at the foramen magnum, less above.

The posterior arch of the atlas does not require baring.

An emissary vein may be encountered in the midline. It is not constant. If present, it may be plugged by a wooden toothpick or by boring into it with an artery clamp.

The self-retaining retractor is now introduced. There are two sizes of detachable blades provided, one for adults and the other for children.

The De Vilbiss trephine (three-eighths of an inch) is applied in the midline, about one inch from the margin of the foramen magnum, and the button of bone removed. With the special dural separators the dura is loosened from the bone and the De Vilbiss bone cutter used to make two lines of incisions or grooves through the bone into the foramen magnum. The dural separator must be constantly used to detach the dura from the bone in advance of the bone cutter. For this reason these separators are made in two sizes, and with a narrow shank to easily pass through the groove in the bone.

The wedge of bone cut loose is about an inch wide at the foramen magnum and a little less at the upper border. Of course the size may vary for each individual and according to complications. The detachment of the bone button is carefully completed, and it is removed.

The dura presents, probably under pressure, bulging into the bone gap.

The occipital sinus (or sinuses) will be seen, if present, showing a blue color through the dura. If the sinus is double, the dura should be incised between them. If single, it should be tied at the upper part and just beyond its bifurcation into the marginal sinuses. The special, full curved, right angled dural needles, right and left, are provided for this purpose.

In dividing the dura, first make a very tiny incision into it, using the fine curved bistoury for this purpose. This is necessary, for should the arachnoid be so closely applied to the dura as to be divided with it, we need to prevent a too sudden escape of the cerebrospinal fluid. Should it be found that the dura has been severed alone, the incision in it should be carried up and down to the limits of the opening in the bone.

The arachnoid will now bulge into the field, unless it has been divided with the dura. The amount of its bulging will

give some idea of the degree of intracranial pressure. The arachnoid is slightly nicked in the middle line and the cerebro-spinal fluid allowed to escape, a specimen being taken for lab-oratory examination. While it escapes slowly a careful watch is maintained upon the blood pressure, pulse and respirations by one especially detailed for this purpose. Syncope may be prevented or lessened by arresting for a moment the flow of the fluid by gentle pressure of the finger. As soon as the excess of fluid has escaped, open the arachnoid for the full extent of the dural opening. The condition in the cerebello-medullary angle should be very carefully investigated. Should there be an exudate about the parts, the lobes of the cerebel-lum should be raised and separated by the "pushers" provided for this purpose, and the patency of the foramen of Magendie assured. It may be necessary to enlarge the opening in the occipital bone. This is easily done by the ordinary rongeurs or by the bone punch devised by the writer. A small wick of rubber or gutta percha tissue is placed within the margins of the dura and arachnoid and left protruding from the wound. The muscles are replaced and held together by two or three plain gut sutures (interrupted). The skin is closed above and below the drain with silkworm gut interrupted sutures. Voluminous dressings are applied, sufficiently thick to fill out the normal hollow between the head and neck.

The patient is handled with care, remembering that the brain stem no longer has its protecting cushion of fluid. The entire operation takes from fifteen to thirty minutes. For further details, the original article should be studied.   *Richards.*

<center>II.—NOSE.</center>

The Submucous Resection of Deflections of the Nasal Septum.

OTTO T. FREER (*Jour. Amer. Med. Asso.,* September, 1912, p. 1127) describes his perfected technic for the performance of the submucous resection of the septum after his method. There is so much detail to the article that the reader interested is referred to the original.   *Richards.*

The Visual Fields in Anterior Nasal Sinusitis.

G. F. C. WALLIS (*Jour. of Laryngology,* October, 1911, p. 511), as a result of further study of the visual fields in

antral, frontal and general sinusitis, reaches the conclusion, twenty-three cases being studied, that peripheral field contraction occurs in fully 90 per cent of all sinus cases, and is usually caused by the action of toxins upon the nerve; the more common is concentric contraction. Central and ring scotoma do not ordinarily result in anterior sinusitis.

Treatment is most beneficial in acute suppurations. The perimeter should always be used in suspected sinusitis. Normal fields help to negative, and contracted fields to confirm the diagnosis. White and green are the best test objects.

Richards.

Some Suggestions in the Methods of Correcting Deflection of the Nasal Septum.

CHARLES W. RICHARDSON (*Jour. Amer. Med. Asso.*, September, 1912, p. 1131) thinks general anesthesia is preferable to local in the performance of the submucous operation, since the shock is much less under general anesthesia, the pain is absolutely annihilated, and the nervous susceptibility of the patient is less disturbed.   *Richards.*

Vibratory Massage in the Treatment of Chronic Nasopharyngitis.

BLEGVAD, Copenhagen (*Zeit. für Laryngologie*, Bd. 5, Heft 1, 1912). Working with a very fine vibratory instrument constructed by C. Nyrop, the instrument maker of Copenhagen, the author claims to have attained very brilliant results in what he calls "the American catarrh."

In a series of one hundred and two cases, fifty-nine were confined to the nasopharynx, thirty-one were combined with nasal catarrh, which was first treated, seven had vasomotor rhinitis, four had respiratory anosmia. In eight cases, three to thirteen treatments were sufficient, in the rest twenty to forty treatments were given to complete a cure.

The results of the treatment were as follows: Fifty-seven were entirely cured, forty-two were very greatly improved. Many of the cases had previously had long treatments with nitrate of silver applications, Lugol's solution, and other local treatments, without results.

The pharynx was previously cocainized, and the mucous membranes were thoroughly massaged for three minutes with a cotton applicator saturated in 2 per cent silver nitrate solu-

tion. In some cases of hay fever the results were especially good, and all the case of vasomotor rhinitis were vastly improved. He claims the good results were obtained because the machine is so constructed as to give a greater number of vibrations per second than any in the market.   *Horn.*

The Results After the Use of Paraffin Injections in Ozena.

LUEBBERS (*Passow's und Schafer's Beiträge,* Vol. 6, No 1, July, 1912), tabulating the results of thirty-one cases of ozena treated in the clinic at Greifswald in the last year and a half, is enthusiastic in the praise of the paraffin therapy. A prelimnary injection of salt solution followed by a one-half per cent injection of novocain was used in order to raise up the mucous membranes and allow a greater deposit of paraffin. The Brunnings syringe and 45° paraffin was used. Several disagreeable accidents occurred in the course of the many injections. There were three cases of septum abscess, one case of general inflammation of the nasal mucosa followed by middle ear inflammation and acute mastoiditis, and one case of the hypertrophic form where all the symptoms were made decidedly worse.

When possible both sides were injected as completely as possible in one sitting. Of the twenty-seven cases which were kept for a year or more under observation, eleven cases were completely relieved of all distressing symptoms and were never obliged to use douches or other treatment again. In ten cases there was a decided improvement, which consisted in the relief from the odor, less crust formation, and decided lessening of the headache and dizziness. In the remaining six cases there was very little or no benefit seen.   *Horn.*

Fibroma of Nose and Nasopharynx.

VIRGINIUS DABNEY (*Laryngoscope,* August, 1912, p. 960) relates an interesting case of fibroma of the nose and nasopharynx. The socalled juvenile sarcoma of the nasopharynx appears between the ages of ten and twenty-five, and is most often on the left side. The tumors most commonly originate from any part of the nasopharyngeal fibrous structure, such as the basilar fibrocartilage, the sphenoid, the adjacent parts of the internal pterygoid process, and the periosteum cover-

ing the sphenoid, and during the age of development are often found to be formed of round cells of an embryonic sarcomatous nature. Before the tumor has reached a considerable size, any hard, tough, gray-pinkish growth should arouse suspicion. From statistics by Delaven, external operations have cured only 59.25 per cent, with a mortality of 25.9 per cent and a recurrence in 15.4 per cent; but intranasal methods gave no recurrence and only a 5 per cent mortality. The author advocates the intranasal operation, followed next in preference by the Doyen operation or one of its modifications.

Richards.

Mucocele of the Frontal Sinus.

GAYLORD C. HALL (*Laryngoscope*, July, 1912, p. 955) reports a case of mucocele of the frontal sinus in which there was marked swelling and protrusion of the left eye and considerable diplopia. Displacement was downwards and slightly inwards. Examination of the nose showed septum deviation on the left above, closely wedging in the middle turbinate, and there had been no history of discharge. Illumination showed both antra to be clear and the frontals extending to the external angular process and above the hair line. Pressure above and backwards at the rim of the orbit revealed an irregular nodular mass, apparently from the roof of the orbit and extending backward as far as could be felt. Exploration was made in the muscle cone behind the eye, but nothing was found, though the optic nerve was followed back to the foramen. Above, a boggy mass without definite outline was felt. After suturing the internal rectus into place and closing the conjunctiva, an incision was made along the brow down to the bone. At the nasal side the knife entered a large cavity and a clear straw colored fluid came out. The soft parts were retracted and the opening in the bone enlarged. The bone was soft, cut easy, and had a water-logged appearance. Examination with the finger found the sinus extending to the external angular process, above and backward about one inch and one-half. The bony roof of the orbit had been entirely absorbed, causing the exophthalmos. The growth felt through the upper lid was the infiltrated edge of this sac. The cavity was emptied of its fluid and stripped of a sticky membrane, and the wound packed and sutured at its inner angle. Dressings changed every other day. The discharge

soon became purulent. Cultures showed a streptococci, and a stock vaccine was given. Soon the discharge decreased and became mucoid in character, and the cavity was injected with bismuth paste. Later a turbinectomy was done. The patient has an occasional discharge through the nose from the sinus, and the eye is again normal.   *Richards.*

Extirpation of Tumors of Vomer Through the Roof of the Mouth.

CHAS. H. MAYO (*Annals of Surgery*, September, 1911, p. 502) reports two cases of malignant disease of the vomer, each with a pear-shaped enlargement of the septum, which completely closed the posterior nares. These growths were removed through the roof of the mouth by the removal of a section of bone one inch long and three-fourths of an inch wide. The soft palate was not severed, a procedure which often complicates the technic of the operation and the after care of the patient.

In preparation for the operation the patient is given thirty to fifty grains of urotropin twenty-four to forty-eight hours preceding the operation, as it undoubtedly aids in preventing meningeal infection. In the two reported cases the patients were given ether to profound anesthesia, following the preliminary hypodermic of 1/150 grain of scopolamin and 1/4 grain of morphin, given two hours before operation, to secure the full effect of the scopolamin.

The resection of the central posterior half of the hard palate is made by midline incision, with preservation of the mucoperiosteum and soft tissues. The position of the patient being the reverse Trendelenburg at this stage of the operation, the head of the table is lowered with the head back in the Rose position, which prevents the blood aspirating into the trachea. The septum is rapidly removed with bone-cutting scissors and curette, and the space packed with gauze. The hemorrhage is quite free during the operation, requiring constant sponging or a sucking apparatus for its removal. The primary gauze pack may be removed within a few minutes and the area of superior attachment of the vomer cauterized with a Paquelin. The nasal space is then packed with benzoated gauze, which is removed on the third day. Accord-

ing to the extent of the disease, some cases may be treated best by immediate suture of the mucoperiosteum, as in a cleft palate operation, while in others it may seem best to maintain the opening, for a time at least, for observation and treatment.   *Richards.*

A Review of Eighty-one Cases of Inflammation of the Frontal Sinuses.

GEORGE L. RICHARDS (*Laryngoscope,* February, 1912, p. 100) found that in eighty-one cases of inflammation of the frontal sinus, 59.2 per cent were acute, 7.5 per cent subacute, and 33.4 per cent chronic. Eighty-five per cent of these complained of pain, either as a headache, severe sharp pain referred to the region of the frontal sinus, or the combination of pain with discharge. Ten had discharge only; one had pain plus exophthalmos, another general anemia and pus absorption, and one only had an opening in the cheek. Forty-one of the acute cases were caused by "grippal cold," in thirty-five cases the cause was unknown. Swelling of the frontonasal duct was direct cause in one. Ethmoiditis, antrum suppuration, and fast automobile riding and dust inhalation are likely causes for a considerable number. In nineteen cases both sinuses were involved; the left sinus alone in twenty-six, and the right in thirty-six. In 91.35 per cent of these cases pus was found, and in the remaining cases there was the presence of gas without pus. Fourteen cases had antrum involvement; three developed otitis media on one side, and one on both sides with profuse discharge. One case was the result of a mastoid from a "grippal cold"; in three there were atrophic rhinitis, in one an external opening, in two nasal spurs, in three septum deviations causing complication, in one involvement of both antra, the right sphenoid, and the ethmoid cells, and in another the involvement of both sphenoids, both antra, and ethmoids. Sixty-five cases were treated intranasally by syringing with some simple sterile saline solution of the Seiler tablet type. Thirty-two of these cases required the removal of the middle turbinate before beginning treatment. Sixteen cases required an external operation with reopening or enlarging of the frontonasal duct and clearing out of the ethmoid cells as far as possible, and after-treatment either by

immediate closure of the external wound or closure at the end of ten days. In six cases the Ogston-Luc operation was done. In two cases the external wound was absolutely maintained until filling up with granulation took place. Two of the Ogston-Luc operations were followed by the Killian operation, and in one case the Killian type was done first. About eighty-three and three-fourths per cent were cured.

Richards.

Submucous Resection of the Nasal Septum.

HANAU W. LOEB (*Jour. Amer. Med. Asso.,* September, 1912, p. 1132) cites the indications and contraindications for the submucous operation, classifying the deviations, spurs and ridges in slight, marked and extreme deflections. The operation is indicated in recurrent acute inflammations of the nose, in due marked and extreme deflections, in acute inflammation of the accessory sinuses when the deflection by interference with nasal drainage is one of the marked predisposing causes, and in hypertrophic rhinitis, and in chronic rhinitis when deviations of the second and third degree are present. In atrophic rhinitis there is some doubt as to whether septal operations are of any value. In chronic inflammation of the accessory sinuses and nasal polypi, hay fever, diseases of the pharynx, larynx, trachea and bronchea, the operation is to be performed whenever there are deflections of any marked degree. The operation, while not curative of these conditions, generally assists in alleviating the symptoms.

In middle ear affections, marked and extreme deflections are to be corrected, and sometimes slight ones. A contraindication is age, the old and the very young not being good subjects for operation. While experience has not yet absolutely determined what influence the removal of a large portion of the septum in the young has on the development of the nose, it is advisable not to operate in children and in young people under eighteen, unless associated conditions involving the sinuses or middle ear seem to make the operation imperative.

Acute sinuses, acute middle ear, pharyngeal diseases, syphilis, tuberculosis and serious general conditions are all contraindications.

Richards.

Acute and Chronic Suppuration of the Nasal Accessory Sinuses.

HERBERT TILLEY (*Lancet,* October, 1911, p. 1179) reviews the entire subject of suppuration of the nasal accessory sinuses and gives the following conclusions:

In acute inflammation of the maxillary antrum, when the antral inflammation complicates a general systemic infection, the inflammation of the mucous membrane is more general and intimate, and the patient's resistance is lower than when the suppuration is caused by a tooth, and hence prognosis is less favorable for rapid recovery in the former than in the latter infection.

If the antral inflammation complicates a constitutional infection, e. g., influenza, in addition to the treatment of the general disease, every endeavor should be made to promote the spontaneous discharge of pus from the antrum. The patient should rest in bed with the infected sinus uppermost, and every hour or two hours an application of equal parts of 10 per cent cocain and adrenalin chlorid should be applied on cotton wool mops to the middle meatus region. This will induce contraction and ischemia of the mucosa and promote the free discharge of pus from the "ostium" in the middle meatus. Hot fomentations to the affected cheek and ten-grain doses of aspirin every four hours will go far to alleviate local pain and discomfort.

When a diseased tooth is the cause of trouble it should be removed, the alveolus perforated, and a temporary alveolar plug inserted. The antrum can then be irrigated twice daily until the inflammation has subsided, when the alveolar passage may be allowed to close.

If bacteriologic examination of the pus should prove that one organism is present or is in great predominance, an autogenous vaccine may assist and prove a valuable adjunct in treatment, but acute cases tend to recover quickly, and a favorable result when vaccine is used may be post hoc, but not entirely propter hoc.

Emphasis must be laid upon the importance of always suspecting sinus suppuration when the patient complains of chronic nasal catarrh, and especially so if pus is contained in the nasal mucus or if the handkerchief is found to be stained by it. A "morning headache" in a patient who has lived in malarial climates might easily be misleading as to antral sup-

puration, but a positive diagnosis can be made by the detection of pus in the middle meatus, by transillumination of the antra, or puncture of the sinus by a fine trocar passed through the inner antral wall.

When nasal polypi and, therefore, ethmoidal inflammation accompany antral suppuration, the prognosis will be less favorable unless radical surgical measures are carried out.

In the majority of chronic cases the best drainage is secured by an opening in the inner wall, but the sinus is first entered through the canine fossa.

The attempt to cure chronic antral suppuration by drainage through the buccal socket of a bicuspid or molar tooth only relieves and not cures the suppuration. The tube or plug, whether it be gold or vulcanite, rarely drains the suppurating cavity, but allows all sorts of organisms and particles of food to gain access to the antrum from the buccal cavity.

In chronic cases of long standing, no procedure short of operation will effect a cure, since the lining mucous membrane has undergone gross degenerative changes. If nasal polypi and the ethmoidal inflammation accompany antral suppuration, the prognosis is still less favorable.

Treatment.—Change of air and internal medication are quite useless. Lavage may be tried for a time, but is usually without result. The diseased mucous membrane must be removed and free permanent drainage provided. This can be provided through the nose by making a large opening in the inner antral wall, after preliminary removal of a sufficient amount of the inferior turbinate bone, or by the same procedure together with the removal of anterior wall of the antrum in the region of the canine fossa—the Caldwell-Luc operation. This is preferable, as it gives access to all the diseased mucous membrane of the antrum. Alveolar drainage in chronic antral inflammation is only to be condemned. The same principles apply to chronic ethmoidal and frontal sinus inflammation, viz., treatment to be efficient must result in free drainage and as thorough removal of diseased structure as is prudent and safe. Operative procedures in these regions should only be performed by those conversant with the anatomy and endowed with a keen tactile sensibility and skilled in nasal surgery. Most cases of acute inflammation of the frontal sinus recover spontaneously since free drainage under normal anatomic conditions is provided for.   *Richards.*

III.—PHARYNX.

Enucleation of the Faucial Tonsil With the Guillotine.

J. K. MILNE DICKIE (*Edin. Med. Jour.*, September, 1912, p. 213) reports fifty cases operated upon with the guillotine after the method of Sluder. In 74 per cent the enucleation was complete. In the remaining cases there was partial success. The success of the operation depends upon the thorough pushing of the tonsil through the ring of the guillotine.

Richards.

Vincent's Angina.

WM. P. WHERRY (*Laryngoscope*, October, 1911, p. 1008), from a study of more than fifty cases of Vincent's angina, concludes that the bacillus fusiformis in its usual activity is not a very virulent organism, that the ground must be prepared by other organisms and by lowered resistance for its invasion. That Vincent's angina is essentially a disease of the crypt of the tonsil induced by poor drainage plus the germ. That the promotion of drainage and the application of 12 per cent silver nitrate is a specific cure. That the usual site of election is the upper angle of the tonsil behind the anterior pillar.

Richards.

Aching Throat.

W. H. KELSOE (*Lancet*, March, 1912, p. 642) describes the aching throat common in people of middle age and associated with flatulent dyspepsia. The pain may possibly be a referred one from the ear, larynx or upper esophagus. Local conditions, although not causing the pain, may accentuate the aching throat. The dyspepsia should be eliminated by diet with abstinence from the use of alcohol and tobacco. In several cases washing out the stomach has relieved the ache. Such drugs as gentian, strychnin, bicarbonate of soda and bismuth have been useful. Operation affords little certainty of relieving the aching throat.

Richards.

Circumcision of the Uvula.

HAROLD HAYS (*Laryngoscope*, July, 1912, p. 967) sprays the region of the soft palate with a 10 per cent solution of cocain and paints the base of the uvula with pure cocain crystals. The entire uvula is anesthetized with an infiltration of 1-10 of a 1 per cent cocain solution. About thirty minims of

solution is used. The uvula increases two or three times its size. The tip is grasped with long artery forceps, after passing it through the oval opening of the author's wedge shaped uvulatome. The desired amount is excised, more mucosa being removed than muscular tissue. If the uvulatome fails to cut way through, it is finished with sharp scissors. The remaining mucous membrane is then brought together over the stump and united with 00 catgut, tying only one knot. The suture is removed on the following day. The throat is gargled the next twenty-four hours with a hydrogen peroxid solution. The author reports five cases so operated, with little inconvenience to the patient from pain and after-treatment.

Richards.

Vincent's Fusiform Bacillus—Experimental Researches.

FRANCIS LASAGUA (*Laryngoscope*, August, 1912, p. 1009) concludes from his experiments with Vincent's fusiform bacillus that the bacillus lives in symbrosis with other microorganisms in the microbic ulcer and together with them determines the initial lesion. It is possible to cultivate and isolate it if present in large numbers. Necrosis can be produced in animals, thus it is evident that in necrotic angina the fusiform bacillus plays the principal part in the development of the disease. But since guinea pigs inoculated with necrotic tissue show more serious and more rapidly developing lesions than those in whom the fusiform bacillus alone was injected, and because in the primary stage of the ulcer Vincent's bacilli are scarce and the other microorganisms numerous, it is concluded that to the latter are due the initial lesions and the increase in the pathogenic power of the bacillus during the course of the ulceration. Vincent's bacillus may be regarded as specific for necrotic angina, since it alone can produce necrosis, and in man pharyngeal ulcers assume necrotic characteristics after its appearance.

Richards.

Tonsillar Hemorrhage—Causes, Prevention and Treatment.

GERHARD HUTCHISON COCKS (*Med. Record*, June, 1912, p. 1032) considers the tonsil operation distinctly a hospital one. Postoperative bleeding is more frequent in adults, in the male sex, and in anemic patients and those subject to hemophilia. Lactate of calcium used previous to operating

often arrests hemorrhage, especially in cases of hemophilia. Postoperative bleeding is often stopped by slapping the face vigorously with a towel wrung out in ice water for a few minutes immediately after the operation. The bleeding usually stops after the appliance of a Mikulicz or Hurd tonsillar hemostat, or, if the bleeding point cannot be found, after pressure of a sponge saturated with gallic and tannic acid for a few minutes. If these measures fail, the author uses Michel's metal clamps, after filling the tonsillar fossa with gauze wet with gallic or tannic acid or powdered thrombokinase (Strong). In cases of hemophilia, the use of blood serum or transfusion is used with good results. *Richards.*

Malignant Tumors of the Tonsil.

JUSTUS MATTHEWS (*Laryngoscope,* May 12, 1912) finds tumors of the tonsil more prevalent in the male sex and appearing, as a rule, after forty years of age. Sarcoma of the tonsil appears encapsulated, the capsule of the tonsil appearing as the capsule of the tumor. Accurate diagnosis is obtained only by clinical signs and symptoms. Carcinoma of the tonsil is usually of an epitheliomatous type. Pain, ulceration and dysphagia are early symptoms in carcinoma, while sarcoma grows to become a large mass without ulceration and is first noticed on account of the size and the difficulty in swallowing. Treatment is indicated by microscopic examination by the methods of frozen section from pieces removed from the growth. Palliative treatment in children is the only one that gives relief. The author reports twenty cases of sarcoma and three of carcinoma treated by tonsillotomy and tonsillectomy, in all of which there was recurrence within two years. One case of carcinoma was free from recurrence for three years after tonsillectomy with the cautery. Jacobson reports a case after this method without recurrence for eleven years. The prognosis of cases treated by splitting the cheek is unfavorable on account of the nature and extensiveness of the growth demanding this operation. The most radical and efficient operation is by lateral and external pharyngotomy in which the entire pharyngeal growth in a block with the cervical gland is removed. X-ray and the use of Coley's toxin give relief from pain and perhaps check the rapidity of the growth. The best medical treatment consists in keeping the diseased areas clean and in reducing the pain. *Richards.*

Tonsillectomy—The Tonsillectome.

JOSEPH A. BECK (*Journal A. M. A.,* January, 1912) claims
that in most cases the tonsils can be removed with a tonsil-
lectome without previous dissection, either with an instrument
or finger. Only under local anesthesia and in cases of exceed-
ingly large tonsils or tonsil stumps does he use dissection first.
His technic is as follows: Patient under general anesthesia,
preferably ether, and in a recumbent position, with head held
firmly by the anesthetist. The tonsillectome is passed behind
the tonsil, going across the tongue from the opposite angle of
the mouth, scooping up the tonsil until it appears in the oral
cavity in the form of a round tumor, behind the anterior pillar.
Holding the instrument firmly in this position, by gentle, steady
pressure the operator feeds the tonsil through the fenestrum
until it disappears under the finger. The snare is unlocked
and the wire drawn down until the tonsil is seen on the inner
side of the fenestrum, everted, forming a cauliflower-like
mass. The tonsil is seized now with a Volsellum forceps, to
prevent it from dropping into the throat when the crushing is
completed. If the patient is now awakening he should be re-
anesthetized while the tonsillectome is in position, and the
tonsil slowly crushed under the mask while etherization is go-
ing on. The tonsil is removed and the capsule is found turned
on itself. On account of the slight bleeding, this method
facilitates an adenoid operation following.   *Richards.*

Enucleation of the Tonsil.

W. S. SYME (*Brit. Med. Journal,* December, 1911, p. 1696)
uses the following method in enucleating imbedded tonsils:
A pair of broad toothed forceps are passed through the loop
of a Krause's nasal snare. The snare is allowed to hang on
the forceps and the tonsil is grasped in its long axis, obtaining
a good grip and drawing it towards the middle line. A non-
cutting, slightly curved elevator, such as Mackenzie's, is
passed through the plica semilunaris, and a sweep is made
downwards, separating the anterior pillar, pressure being at
the same time applied by the elevator to draw the tonsil out
of its bed. The posterior pillar is then treated in the same
way. The upper part of the tonsil then emerges from between
the pillars, and the snare is passed over it and drawn tight, the
point of the barrel being pressed against the lower end of the

anterior pillar. To avoid hemorrhage the snare is left on while the other tonsil is removed.

Thomas Guthrie operates after the Sluder method, the tonsil being drawn by the ring of the guillotine upwards and forwards over a bony prominence on the inferior maxilla, called by Sluder the eminentia alveolaris, which in turn pushes it through the ring. The tonsil is still further pressed through the ring by the operator's finger, applied to the anterior pillar of the fauces and the adjacent part of the soft palate. He prefers the left lateral position, grasping the guillotine with the right hand for the right tonsil, and with the left hand for the left. *Richards.*

A Method of Enucleation of the Tonsil.

CHARLES W. M. HOPE (*Brit. Med. Journal,* March, 1912, p. 542) describes his tonsil enucleation with the patient in a supine position and head slightly retracted. When the patient is thoroughly anesthetized the tongue is drawn out of the mouth by a pair of tenaculum and held by an assistant   The left anterior pillar of the fauces, low down, is then grasped with a pair of straight forceps held in the operator's right hand and pulled forwards and outwards away from the tonsil. By means of a closed pair of forceps, used as dissector and held in the left hand, a hole is torn through the mucous membrane lying between the tonsil and portion of the anterior pillar held by the right hand forceps. The white pearly capsule appears and the opening is deepened about one-half inch. With a steady circular motion the dissecting forceps are swept up the front of the tonsil, across the upper pole, and down its posterior side, separating it from its anterior upper and posterior attachments. The right anterior faucial pillar is then grasped with the left hand pair of forceps and the dissection made as before with the right hand pair of forceps. The tongue is then released, forceps put aside, and the separation of the deep surfaces of the tonsils is completed with the index finger. The tonsils are stripped down easily to the lingual prolongation and hang free in the pharynx, attached only at the lower pole. The patient is turned on his right side to drain out the blood, and enucleation completed by passing a guillotine over each tonsil and dividing the small pedicle at the lower end. This method of complete enucleation with capsule intact, plus the curetting of adenoids, is done in from forty to sixty seconds.

Richards.

The Operation of Tonsillectomy as Performed in Ancon Hospital.

HOWARD V. DUTROW (*Laryngoscope*, May, 1912, p. 753) considers tonsillectomy a hospital operation. The operation in children and nervous adults is done under general and profound anesthesia, ether preferred. Local anesthesia is permitted in children above fourteen or fifteen years of age, and in adults who have good control of their nervous temperament. He gives the principles of his operation as follows:

1. Patient under deep anesthesia and lying on the right side, with head lowered fifteen or twenty degrees.

2. Swab the fauces clear of mucus.

3. Grasp the tonsil with forceps such as Ballenger's or Burrows', with one jaw in the base of the tonsil and the other as far as possible in the supratonsillar fossa.

4. Separate with knife or scissors the mucous membrane from the tonsil, anteriorly, posteriorly, and superiorly, and dissect back beyond the margin of the capsule.

5. Bring the tonsil forward into the fauces and snare, care being taken to place the wire in line of incision and outside of the capsule. Prevent catching the uvula and faucial pillars in the wire loop.

6. Inspect tonsil cavity and see if enucleation is complete. If any portion of the superior lobe remains, enucleate in same manner.

7. Apply a 1 to 1000 solution of adrenalin to control bleeding.

8. To control hemorrhage use some hemostatic agent such as a strong solution of silver nitrate, adrenalin, etc., together with pressure and an ice-cap to each side of the neck. Seize the bleeding points with artery forceps and tie if these measures fail.

9. Keep the patient in bed twenty-four to forty-eight hours. Gargle the throat with a dilute Dobell's solution.

10. Paint the tonsillar fossæ once or twice daily with tincture of iodin, which lessens the trauma of the uvula and facial pillars and facilitates the healing.   *Richards.*

An Improved Method of Using the Tonsil Guillotine.

A. H. PROCTOR (*Indian Medical Gazette*, January, 1912, p. 19) describes his technic as follows: The patient is lying on his right side, the operator stands opposite his chest, looking

towards his head, with the light shining well into the patient's mouth.

A Mackenzie or similar guillotine is passed into the mouth, and using it in the same way as a Fränkel's tongue depressor the tongue is pressed down till the lower pole of the tonsil is seen. The ring is then pressed firmly upwards so as to engage the lower pole of the tonsil.

Second stage.—The hand is now pronated so that the shaft becomes vertically placed, the right edge looking towards the roof of the mouth and the left resting on the tongue. At the same time the handle is carried over from the middle line to the opposite angle of the mouth so that the shaft runs obliquely from the left angle of the mouth to the posterior pillar of the fauces, against which it now rests. By now continuing this latter motion the tonsil and interior pillar of the fauces can be levered or lifted forward towards the teeth till anterior pillar is stretched taut across the ring of the guillotine.

Third stage.—To the outer side of the margin of the anterior pillar will be seen a rounded elevation caused by the tonsil. If now the thumb of the left hand be pressed on this the tonsil can be pressed through the ring of the guillotine. It will be felt to engage in the ring, and if the pressure be continued the anterior pillar becomes everted and thereby lifted up, so that the blade passes just under its margin. At this moment the blade is gradually driven home, and as soon as the attachment to the anterior pillar is severed the pronation of the hand is continued. By the time the hand is completely pronated and the under surface of the shaft looks towa 's the roof of the mouth, the blade has gone right home. The tonsil comes out resting on the under surface of the blade.

The left tonsil is removed by a similar procedure, but in this case the patient is best on his back, the operator standing at his head, looking towards the patient's feet. The tonsils should be at once examined to see if their capsules are complete. Any small portion that may have been left can be removed in the same way.   *Richards.*

Tonsil Enucleation With the Guillotine.

WHILLIS AND PYBUS (*British Med. Journal,* November, 1911, p. 1402; *Lancet,* September 17, 1910), in two articles, describe their experience in enucleation of the tonsils with

the guillotine after the manner of Sluder given in the
Annual of last year. In their first series of 200 cases they
were able to enucleate 42 per cent complete in their capsule in
one piece. In a more recent series of 100 cases, 74 per cent
were enucleated complete. Their usual anesthesia is ethyl chlo-
rid, and a guillotine modified after that of Mackenzie is used.
Various sizes of these should be at hand. When anesthetized
the patient is turned partly over onto the right side, the head
lying on its right side on a level with or slightly above the
trunk, so that the cheek pouch is on a lower level than the
fauces and that blood may readily collect and run out of the
mouth. The gag is then opened. The guillotine, with the
shaft specially thickened, is first used as a tongue depressor and
the lower tonsil seen. The operator stands facing the patient's
head, on the right side. The guillotine being held in the right
hand, the ring is passed under the lower border of the tonsil,
which is pressed upwards towards the soft palate. The left
index finger is then placed on the outer part of the anterior
pillar of the fauces and presses the tonsil into the ring. At
this time the blade is gradually pressed home with the thumb
of the right hand. It enters between the tonsil and the anterior
pillar, cutting the mucous membrane connecting the two.
While cutting, the hand is gradually pronated, so that the
under surface of the guillotine looks inwardly and finally
upwards, the tonsils being separated from the pharyngeal wall
during this maneuver; the final cut severs the mucous mem-
brane connecting it to the posterior pillar. The tonsil is then
lifted out on the under surface of the guillotine, which is now
uppermost. The right tonsil is removed first. To remove the
upper tonsil the patient is rolled back so that the head lies in
the dorsal position. The operator now passes to the side of
the patient, the guillotine is again inserted, and the ring passed
below and behind the tonsil, which is pressed upwards towards
the soft palate, the left index finger being again used to force
the tonsil into the ring. The connections are cut through as
above described, the hand being meanwhile pronated and the
tonsil removed on the under surface of the guillotine, which
has become uppermost. In less than half the cases the tonsil
can be felt to slip into the ring, and in these instances it fre-
quently comes out entire. In others it will not wholly engage,
and one can say definitely that two or more attempts will be
necessary to remove the whole tonsil.   *Richards.*

Radical Tonsil Operation.

S. TENZER (*Wien. klin. Woch.*, January, 1912, p. 122) has performed the radical operation for the tonsil for two years, but only in cases of adults or older children in which relatively small tonsils have been the cause of rapid angina or peritonsillar abscesses. In children the old methods are still used. He then states, while admitting that a certain number of tonsillotomies in children are followed by trouble afterwards, he did not find these sufficient in number to require tonsillectomy in every child. For the last year or so Tenzer has used the method of Sluder in upwards of 1,000 tonsillectomies, and as a rule without narcosis. He finds that the time required is only a little longer than the ordinary tonsillotomy. About ten seconds can be reckoned for each tonsil. But when the operation is done without anesthesia it is much more painful than the simple tonsillotomy, as the instrument is used with considerable force and pushed pretty hard against the upper jaw. Local anesthesia lessens the amount of pain but does not take it entirely away, as it is not the cutting but the pushing of the instrument which causes the pain. The immediate bleeding after the operation is more than after tonsillotomy, but in this series of 1,000 tonsillectomies there has been no after-bleeding. The child is kept still for a little while after the operation. Reexamination at the time finds the bleeding usually to be stopped. Only in one case of an anemic child was there any bleeding, but this was in a case in which the operation should not have been done, and the bleeding would have been just as great in tonsillotomy.

In cases of adults, however, the result as to the bleeding is quite different. Four or five cases bled severely. The source of the bleeding was several times in the pillars, several times in the floor of the tonsil fossa, and several times from the arteries in the pharynx muscles which supply the tonsils. On account of the gagging and wretching, it is difficult in these cases to find the source of the bleeding. The arteries are grasped with long forceps and ligated or a gauze sponge compressed with Mikulicz forceps.

The bleeding stopped in every case on the same day. In reference to the after-reaction, the borders of the pillars did not show any signs of bad handling which is often seen after the socalled surgical tonsillectomy. The operation is therefore

to be recommended rather than tonsillotomy, since its results are so much better. A certain trick is necessary in order to remove the tonsil in this manner, and considerable practice is required in order to completely remove the tonsil, and there are forms of flat tonsils which cannot be removed in this manner.

In regard to the question whether children shall be operated by radical removal, the author says that several years must elapse and a sufficient number of cases collected before this question can be determined.   *Richards.*

Tonsil Removal—Quinin Anesthesia.

SHEEDY (*Medical Record*, October, 1911). Tonsillectomy should be a hospital operation. Sheedy reports four cases of death following the use of cocain and adrenalin solutions. In the first case 1/12 of a grain of cocain and from 8 to 10 minims of adrenalin solution had been given to a man of thirty in apparently good health. His tonsils were removed without hemorrhage. The operation was followed by marked prostration and shock. Patient placed in recumbent position, restoratives and artificial respiration applied, no result. At autopsy right auricle swollen and full of blood. Thymus enlarged.

Second case: Man twenty-five, in good health except for indisposition due to tonsillitis. One dram of solution made up of 1 grain of cocain and 1 dram of 1 to 1000 adrenalin chlorid to 2 ounces of normal salt solution was injected in and around each tonsil. Patient became faint, broke into profuse perspiration, restoratives applied. In a few minutes put in upright position and tonsils removed. No hemorrhage. On account of shock was kept in room adjoining operating room for more than an hour, then started for home, but when about a block away fell in the street and before assistance arrived was dead. Autopsy: All important organs apparently normal.

Case three: Man about forty-four, in good health except for an attack of kidney trouble following scarlet fever when a boy. One dram of solution of cocain containing 1 grain to the ounce with from 7 to 10 minims of adrenalin chlorid was injected in and around the tonsil. As part of the injection seemed to escape, in about fifteen minutes the injection was renewed. In three minutes' time the patient became deathly pale and pulseless. Respiration slow and labored, improved

slightly under hypodermic of sulphate of strychnin, 1/30 of
a grain. The head was supported by an assistant and tonsil
enucleated. There was marked weakness and prostration.
Patient placed in bed, heart soon ceased to beat, restoratives
applied, no result. No autopsy.

Case four: Young man of twenty-five or thirty, slightly
nervous, deviated septum and large tonsil, otherwise in good
health. Primary anesthesia with 3 parts ether and 2 of chloro-
form; 15 minims of 1 to 1000 adrenalin chlorid solution was
injected, divided into three doses. One-half minute after the
last injection the patient's skin blanched as white as possible.
Respiration deepened and slowly ceased. Restoratives ap-
plied without result. No operation had been performed, there
was no autopsy.

These cases show the great danger in the use of cocain and
adrenalin solutions when injected into the soft tissue surround-
ing the tonsils. When local anesthesia is desired, let the appli-
cation of cocain be limited to the surface and not injected.

To prevent pain and still to attain a sufficient degree of local
anesthesia for the operation, the author suggests the use of a
5 per cent solution of bisulphate of quinin. There has been no
pain in the cases in which the solution was deposited outside
the capsule of the tonsil and into the tissue forming its bed.
One-half dram of solution is introduced by means of a one
ounce piston syringe outside of the border of the anterior pil-
lar, and the same amount at a point opposite, between the cap-
sule and posterior pillar. Enucleation can begin as soon as the
solution is injected, there being no occasion for waiting, as
when cocain is used. No preparation of the patient is neces-
sary. The patient sits upright in the chair, with the head
properly supported.

The technic does not differ from that of other authors.
Traction is maintained on the tonsil and the caution given not
to cut through the capsule, otherwise the proper line of cleav-
age will be difficult to find. The tonsil is properly everted and
the wire snare loop used. The snare takes the plane of least
resistance, and the capsule is shelled out from its bed. In
children the use of ether anesthesia is advised.   *Richards.*

IV.—LARYNX.

Singer's Nodule Removed by Vocal Treatment.

F. VICTOR LAURENT (*Jour. Amer. Med. Asso.*, September 30, 1911) cured a case of corditis nodosa by teaching of the proper use of the voice. He uses low tones, pitched in the register below that in which the patient is accustomed to talk.

Richards.

Chronic Laryngeal Stenosis—Treatment by Prolonged Intubation.

HOMER DUPUY AND L. DE POORTER (*Jour. Am. Med. Asso.*, September, 1912) have devised some special tubes with a low retaining swell so placed that it impinges on that portion of the subglottic and tracheal areas which are a usual seat of the constriction. This tube reduces the possibility of autoextubations, and effectively carries out the essential principles of dilatation. Persistent laryngeal stenosis in children has for its distinctive features exudative and inflammatory changes with a narrowing of the respiratory lumen in the subglottic and other tracheal areas, and while a pathologic entity in itself, it must be considered as the end result of some active inflammatory process which may be of diphtheritic origin. In the present state of our knowledge, prolonged intubation offers the best prospects of success in overcoming the stenosis.

Richards.

Intrinsic Cancer of Larynx.

ST. CLAIR THOMPSON (*Brit. Med. Journal*, February, 1912). In ten cases of intrinsic cancer of the larynx operated upon by laryngofissure, cure was present in seven cases for a period of from fifteen months to seven and a half years. One died three years later from cancer on the tongue with no recurrent growth in the larynx. The second died fifteen months later from another cause, and the third from local recurrence. All the cases were males of from forty-three to sixty-eight years of age, and one was nearly seventy. Huskiness was the principal diagnostic symptom. In no case did thyrotomy disprove the diagnosis. In no case was there any enlargement of the cervical glands, and in one case a gland on the opposite side of the neck was enlarged. Microscopic examination was done only in three cases, since the cancer usually appears as an infiltration of the cord, making removal impossible. Any attempt at removal tends to extend the disease and alters the local appearance so that inspection is difficult, besides the removal

of an unsatisfactory portion of the cord makes diagnosis misleading. Impaired mobility of the cords was present in 50 per cent of the cases and is a valuable sign in diagnosis. In two cases granuloma appeared on the cords two or three months after the operation.   *Richards.*

The Ambulatory Treatment of Laryngeal Tuberculosis.

ARTHUR MEYER (*Berlin. Zeit. für Laryngologie,* Bd. 5, Heft 1, 1912). This ever present question is discussed in a slightly more optimistic vein by the author, who in twelve cases which he has been able to follow, has secured ten cures.

Without question the all important thing is the lung condition, and any methods of treating laryngeal tuberculosis which does not give this factor first place must be valueless. The one remedy which is most to be relied upon is tuberculin, skillfully administered. Of the local measures Meyers gives the choice to the flat burners, considering the deep galvanopunctures as not so valuable, and the deep destructive burnings with the porcelain tip as too severe for ambulatory cases.

Contraindications he tabulates as follows:

A. For the use of tuberculin:

1. Severe or extensive lung involvement.
2. High fever.
3. Bad social conditions.
4. Intestinal complications.
5. Cachexia.
6. Tendency to hemoptysis.

B. For active local therapy:

1. Same as 1 to 5.
2. Extensive or rapidly progressive disease of the entrance of the larynx or the pharynx.
3. Acute miliary processes in larynx.
4. Extensive involvement of the trachea.

He has seen very good results follow the amputation of the epiglottis and considers as indications:

1. Where the process is entirely confined to the epiglottis.
2. In severe dysphagia, where the origin is in the epiglottis.
3. In epiglottic involvement, without dysphagia, when the lungs are only slightly involved.

He considers it practically impossible in this class of cases to require absolute silence, and thinks that the whispered voice

is more irritating to the larynx than the spoken.   He orders
these cases to speak as little as possible.   *Horn.*

Suspension Laryngoscopy.

KILLIAN, Berlin (*Archiv. für Laryngologie,* Vol. 26, No. 2),
in a long article, gives, for the first time, the details of what
he evidently believes to be one of his most important contribu-
tions to the subject of bronchoscopic methods.   The problem
which presented itself was to devise some method by which
intralaryngeal procedures could be carried out as thoroughly
and with as little limitations as to time as is now the laryngo-
fissure.

After many experiments, which are fully described, he final-
ly evolved an apparatus which is fully described in this article.
The procedure is about as follows: The patient is placed on
his back with the head over the end of the table.   A spatula,
attached to a long arm, the shape of which is like a Kirsteins
spatula, is hooked over the epiglottis, a tooth piece is brought
under the upper teeth, to keep the apparatus from slipping
out, and the long hook is attached to an upright which has been
screwed to the table.   The weight of the head thus rests on
the base of the tongue and epiglottis, the reflexes are abolished
and the interior of the larynx, the arytenoids and the subglottic
space can be seen with great clearness.   Both hands are free
to operate with, the source of light being a recently modified
Kirsteins head lamp.

Procedures which so far have been principally removal of
papillomatoma in children and the very thorough curettement
of tuberculous ulcers in adults, have been carried out with
great ease.   Some of the sittings have lasted an hour.   Anes-
thesia generally local, preceded in most cases by scopolamin-
morphin, was used.   *Horn.*

V.—MISCELLANEOUS.

A Case of Scleroma.

SAMUEL IGLAUER (*Laryngoscope,* August. 1912, p. 1012)
reports rhinoscleroma to be rare in the United States.   The
disease is characterized by granulomatous infiltration in the
mucous membrane of the nose or of the naso- or oropharynx;
the granulomata gradually transform in the connective tissue,
causing marked stenosis of the affected parts.   The author
cites one case of rhinoscleroma.   Excellent results were ob-

tained from the use of radiography (Zwillinger) and the prognosis under this treatment is more hopeful.   *Richards.*

The Black Hairy Tongue.

WILLIAM LESLIE CARTER (*Laryngoscope*, August, 1912, p. 1027) finds the black, hairy tongue more frequent in men than in women and that the majority of the cases are excessive tobacco users. The disease may be acute or chronic, lasting from a few days to fifteen years. The disease occurs mostly in middle aged people. The author believes the affection is due to a local nutritive disturbance, and the growth of the filiform papillæ is similar to that of papillomata in other parts of the body. He reports two cases.   *Richards.*

The Eye and Accessory Sinus Disease.

FRANK BRAWLEY (*Laryngoscope*, October, 1911, p. 1013) claims that asthenopic symptoms due to sinus disease are usually reflex in nature, but may also be due to a toxic process or stasis in the orbital circulation resulting from the circulatory disturbances within the diseased sinuses. Diseased ethmoidal cells are found to be more often the source of ocular trouble than the frontal and maxillary sinuses. The X-ray and vacuum methods of diagnosis aid in finding sinuses causing ocular inflammations. The difficulty to differentiate between orbital disease of sinus origin and that due to new growths is eliminated by nasal findings, skiagraphs, temperature and differential blood count.   *Richards.*

Cyst at Base of Tongue.

EDGAR A. FORSYTH (*Laryngoscope,* December, 1911). Cysts of the dorsal aspect of the tongue are uncommon, but the most frequent are the mucous cyst, blood cyst, cysticercus cellulosæ, echinococcus, and chronic abscess. Mucous cysts are found behind the middle of the tongue, and are seldom larger than a hazel nut. They occur more frequently in adults and are difficult to diagnose. Cases of cysticercus cellulosæ are exceedingly rare, only one case has been recorded. Echinococcus are uncommon, occur mostly in grown people, and are situated in the muscular substance of the tongue. Chronic abscesses are common in the dorsum of the tongue, in front of the circumvallate papillæ, while mucous cysts are found behind the papillæ. The author concludes that complete removal of cysts is

advisable, preventing recurrence; that incision, giving only temporary relief, admits a possibility of recurrence.

Richards.

A Case of Lingual Thyroid.

GEORGE FETTEROLF (*Laryngoscope,* August, 1912, p. 1015) reports a case of lingual thyroid in a man of thirty-four who was of a tubercular nature. Until examination with laryngoscope he was unconscious of the existing tumor. The growth was hemispheric in shape and situated far back on the tongue, its posterior termination ending at the supralingual portion of the epiglottis, while the anterior margin was about on a line with the anterior pillar of the fauces. It was the color of the deepest red of the faucial pillars, smooth to the touch and had a feeling of fluctuation. After questioning its nature as a malignant or benign growth, cyst, chronic tubercular abscess and accessory thyroid, it was diagnosed as accessory thyroid, since there was a quantity of large engorged veins over the surface of the growth. Owing to the patient's general condition, operation was not considered. Out of sixty-seven cases reported by various operators, only three have died from this tumor, and extirpation is only resorted to when the respiratory or alimentary tracts are in danger. *Richards.*

Acute Inflammation of the Thyroid Gland.

OTTO J. STEIN (*Laryngoscope,* August, 1912, p. 1021) claims inflammation of the thyroid gland to be an endemic disease particularly affecting females between the age of twenty and forty. Sixty per cent of the thyroid inflammations suppurate and twenty per cent of these die. A simple inflammation is never fatal. Inflammation usually takes place in an existing large gland, at times occurs in the course of pneumonia, influenza, eruptive diseases of childhood, typhoid, diphtheria, erysipelas, mumps, malaria, rheumatism, tonsillitis, lues and tuberculosis. It has occurred after an injury or operation on the neck. The symptoms of an acute inflammation of the thyroid gland will vary somewhat with the absence or presence of pus. Both varieties have the usual symptoms of an ordinary febrile disease, like chilliness, high temperature, dry and hot skin, anorexia, constipation, maybe nausea and vomiting, and perhaps some headache. It is only when the focal symptoms begin to appear following one of these diseases that suspicion is aroused. *Richards.*

INDEX OF AUTHORS.

INDEX OF TITLES.

1101 *1107*

Annals of
Otology, Rhinology and Laryngology

1145 dooskup for this meetg *R*

CONTENTS

CONTENTS—Continued from Second Cover Page

125
100
35
550

35
1380

54